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RECOGNITION OF PRECANCEROUS LESIONS OF THE ORAL CAVITY

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CANCER IS one of the most insidious and formidable diseases confronting mankind today. With regard to the dentist's responsibility in the diagnosis and prevention of malignant lesions of the mouth, national statistics tell us that 5.2 per cent of all fatal cancers in men, and 1.2 per cent of all such lesions in women, originate in the oral cavity.[1] Of 207 confirmed malignancies in the Tumor Clinic of Huntington Memorial Hospital, Pasadena, 12.5 per cent were within the field of oral surgery.

In the course of daily routine, dentists have an opportunity to examine the oral cavity in its entirety and to observe early changes in the texture of the mucous membrane, long before the development of malignant degeneration.

Once malignant changes have begun, the process is wont to be rapid, and after a few months have elapsed even the ever-improving methods of modern treatment will lead to a cure in only a fair percentage of cases. In cancer of the tongue, the most favorable rate of recovery reported has been 66.6 per cent for cases in which treatment was initiated within less than two months after appearance of first symptoms.[2] Generally speaking, the chance for a cure of almost every type of intra-oral cancer is at least twice as great if aggressive treatment is initiated at an early date.[1] Figures demonstrate beyond any doubt that delay in the diagnosis of an existing malignant lesion may make its successful eradication impossible.

Read at the twenty-ninth annual meeting of the American Society of Oral Surgeons, Boston, July 31, 1947.
Photographs for Figures 4 and 5 were furnished by George S. Sharp. Photographs for Figures 6 (left) and 8 were furnished by Walter F. Schwartz.

1. Martin, H. E., Mouth Cancer and the Dentist. *J.A.D.A.* 33:845 (July 1) 1946.
2. Sharp, G. S., and Spickerman, H. D., Cancer of the Tongue. *Am. J. Roentgenol.* 57:181 (February) 1947.

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On the basis of case histories from a leading cancer hospital, it has been established that over 31 per cent of patients with cancer in different parts of the oral cavity consult a dentist before they ask for medical advice. However, it was concluded that the existence of cancer was recognized by the dentist in only 38 per cent of all cases, most frequently (50 per cent) when it appeared in the buccal mucosa, and least often (31 per cent) when the lesion was located in the gingiva.[1] This makes it clear that dentists are likely to treat a lesion of the gingiva by one of the methods well known to the profession, but that the farther away from the teeth the lesion appears, the less certain is it recognized as one of the nonmalignant conditions which can be dealt with in a routine manner.

Diagnosis of Lesions

It therefore follows that it is necessary for the dentist to bend every effort to sharpen his discernment between cancer and other pathologic conditions involving the oral mucosa. There can be little doubt that this distinction should be stressed increasingly in undergraduate and postgraduate training. It goes without saying, however, that the greatest improvement in that direction cannot change the fact that no definite recognition of a malignant condition is possible on clinical evidence alone, and that a conclusive diagnosis can be expected only after microscopic examination of a tissue specimen. It may be that in the past we have been reluctant to voice a suspicion of malignancy, fearful of producing an undesirable effect on the patient's mind; but in the light of better understanding there is no room for such hesitation. Removal of a small specimen of tissue and the laboratory examination that follows is a simple procedure and one that should be used more freely by our profession.

The conditions usually present preceding the appearance of cancer may be divided into (1) malignant degeneration in almost any kind of benign tumor of long standing; (2) mechanical and chemical irritation and certain conditions of dental and oral cleanliness, and (3) a number of physiologic changes within the oral mucosa.

Clinical observation and experimental research have established the fact that a number of conditions or changes have a bearing on the ultimate development of cancer. These data retain their importance even though the strictly local origin of a cancerous lesion has of late become doubtful,[3] and it is assumed that an ill-defined diathesis[2] or a constitutional factor may be of etiologic importance.

Yet, in regard to all these conditions, "it should be clearly understood that a precancerous lesion is not the cause of cancer, but rather that precancer is a prodromal stage or prodromal symptom which indicates that the chance for development of cancer is far greater in a given case or at a given site, than in the normal."[4]

The use of the term precancerous lesions has been the subject of extended controversy. There can hardly be any doubt that the term is being misused when it is applied to conditions in which at least one typical cancer cell has been demonstrated by histopathologic examination, although neither invasion of the basement membrane nor lymphatic permeation has taken place. Different terms have been proposed to distinguish such pre-invasive cancer from frank cancer on the one hand, and from precancerous lesions on the other.

Precancer as it is used here does not refer to any lesions in which histologically typical cancer characteristics are to be found even in a single epithelial cell; neither is the term employed to indicate any inevitability that the lesion will become malig-

3. Quick, D., Leukoplakia and Allied Mouth Conditions. *Canad. M. A. J.* 40:234 (March) 1939.
4. Martin, H. E., and Koop, C. E., Precancerous Mouth Lesions of Avitaminosis B. *Am. J. Surg.* 57:195 (August) 1942.

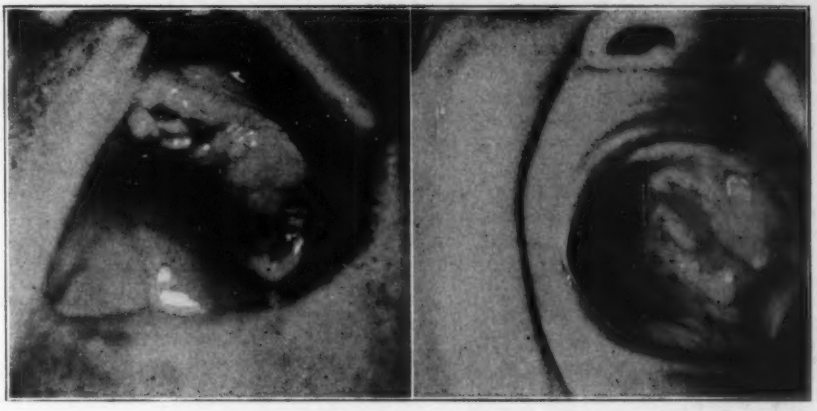

Fig. 1.—Left: Ossifying fibroma. Right: Fibrochondroma

nant. Precancerous lesions are all those in which, after careful scrutiny, including microscopic examination, there is found to exist no evidence that the growth will eventually become malignant but in which, on the basis of past experience, we must assume that in a certain percentage of cases carcinoma will develop in the course of time, and in which, therefore, a greater likelihood of such an occurrence exists in the individual case than if only healthy tissue were present.

Malignant Degeneration

The first group of precancerous lesions includes the benign tumors of the oral cavity which, like tumors in any other part of the human body, may in due time undergo malignant degeneration. This is true even of simple, chronic or inflammatory ulcers, the latter caused by Vincent's organisms or trauma from rough or jagged surfaces of teeth, and ulcers of tuberculous or syphilitic origin. Cancer also may arise from warty lesions and from the lumpy tumors that follow mycotic infections, especially blastomycosis and actinomycosis. Chronic dental abscess may lead to the formation of a radicular cyst which, if unattended, may finally assume a malignant character. Infection or irritation between the teeth may produce the fibrous tumor of epulis which resembles cancer and later occasionally becomes cancerous.[5] All the more common types of benign tumors are occasionally encountered in the oral cavity: angioma, hemangioma as well as lymphangioma; fibroma; granuloma; adenoma; papilloma; myxoma; melanoma and pigmented nevi. More rarely observed, but typical in their appearance within the oral cavity, are mixed tumors of the salivary glands and giant-cell tumors of the mandible.

By this bare enumeration, attention is drawn to the variety of tumor processes which may originate within the oral cavity. Sometimes a mild but prolonged irritation leads to hypertrophic fibrosis in the submucosa which may turn into a true fibroma. Or a mere varicosity of a small group of vessels may change into a submucous angioma. Once started, all such lesions are usually progressive and in the end may either themselves degenerate toward malignancy or give rise to the development

5. Pfahler, G. E., Prevention and Early Diagnosis of Mouth Cancer. *Bull. Am. Soc. Control Cancer* 22:5 (December) 1940.

of a cancerous growth. Therefore, one should regard with suspicion any persistent chronic swelling in the lips or in any part of the buccal or nasopharyngeal mucosa.

Clinically as well as microscopically it is impossible to determine the exact moment at which a tumor ceases to be benign and becomes cancerous. But malignancy should be strongly suspected whenever sudden changes in the physical appearance of a hitherto benign tumor occur; pigmentation may increase noticeably, or rapid growth may set in, or profuse scaling or excoriation can be observed, or bleeding ensues, or cracks, ulceration, lumps or an induration develop.[6] If one or more of these symptoms appear in a tumorous growth, cauterization is decidedly contraindicated, as by this method of treatment the spread of carcinoma could be stimulated. The only safe procedure is to obtain a definite diagnosis based on microscopic examination of a tissue specimen.

Chronic Irritation

A second group of precancerous lesions of the oral cavity is caused by conditions producing chronic irritation through various mechanisms, which from past experience are suspected of contributing toward the development of malignancy.

Chronic irritation may result from a number of different causes or agents. Among the chemical irritants tobacco in its different forms is probably the most important factor. Excessive smoking has formerly been considered to be directly responsible for the appearance of leukoplakia. More recent research, however, tends to interpret leukoplakia and similar conditions as forms of degenerative changes within the mucosa which, although not caused by the use of tobacco, are undoubtedly aggravated in the mouth of the smoker. The specific irritation caused by tobacco has been attributed to a carcinogenic compound (benzpyrene)[7] or to presence in the finished product of arsenic used for spraying during cultivation.[8] There can be little doubt that the stronger forms of tobacco are more likely to produce irritation.[9] Yet, in spite

6. Simpson, F. E., Precancer of the Lip. *Illinois M. J.* 79:459 (June) 1941.
7. Grace, E. J., Influence of Carcinogenic Compound (Benzpyrene) in Cancer of Oral Cavity. *J.A.D.A.* 30: 1358 (September 1) 1943.
8. Hollander, L., Shelton, J. M., and Shonfield, L., Practical Considerations with Regard to Diagnosis and Treatment of Intra-Oral Cancer. *J.A.D.A.* 28:572 (April) 1941.
9. Somervell, T. H., Recent Advances in Treatment of Carcinoma of the Mouth and Jaw. *Brit. J. Surg.* 32:35 (July) 1944.

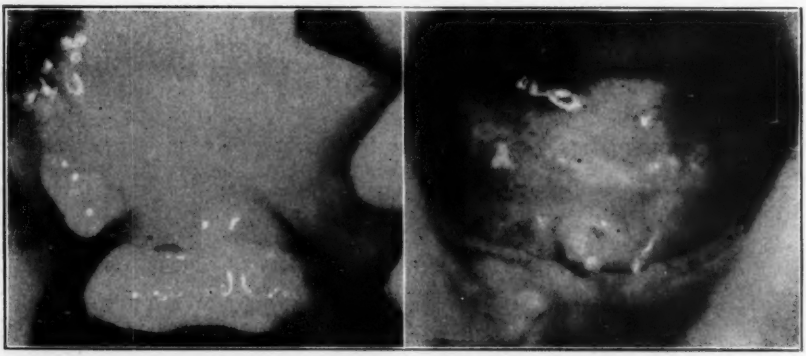

Fig. 2.—Left: Irritative fibro-epithelial hyperplasia. Right: Reactive hyperplasia of floor of mouth with marked leukoplakia. Trauma caused by ill-fitting lower denture impinging on genial tubercles

Fig. 3.—Early fibro-epithelial hyperplasia caused by ill-fitting denture

of this evidence, it is extremely unlikely that tobacco alone will produce cancer. Another author assumes that of the three principal etiologic factors of epidermoid carcinoma of the buccal mucosa, syphilis, tobacco, and dirty, broken and infected teeth, at least two factors must operate over an extended period of time in order to produce malignancy.[10] Chemical irritation may be induced by other agents such as the dyes and pigments in lipsticks, various cosmetics, highly seasoned foods and strong proprietary mouthwashes. Actinic irritation may be traced to excessive irradiation by either roentgen rays or radium. Among the etiologic factors of cancer of the lip, constant exposure to the sun plays an important part.

Another group of irritants is of a mechanical nature, and gives rise to the various forms of traumatic lesions. Wounds may be caused by occupational hazards or habits. The oral mucosa may become injured when nails, pins, or tarred needles are held in the mouth, or through biting thread. In other instances of oral cancer, the initial trauma may be caused by hot steel splinters, hot tar, broken glass, or severe burns, brought on either by accident or by removal of growths on the lip by cautery.[11] Cancer may arise in scars formed after previous injury, or may be traced to biting of the lips, or cuts with the razor.

Various dental factors are of greatest importance as exciting causes of traumatic lesions from which malignant growths may develop. In cancer of the tongue, about half of the lesions occur along the lateral margins, a region in which trauma caused by tearing, or erosion from jagged, broken, worn or rough teeth is frequently observed.[2]

The connection between a single injury from a sharp tooth and the development of carcinoma at the precise point of injury to the oral mucosa is so obvious that since the days of Hippocrates dental disorders have been regarded as directly carcinogenic. Yet, in the average case, the presence of more than one irritant can be demonstrated, even though therapeutic tests may be required in order to determine the relative etiologic significance of various factors producing similar reactions within the oral mucosa.[4] It has been pointed out that some degree of dental disorder is encountered

10. Lund, C. C., Epidermoid Carcinoma of the Buccal Mucosa. *Surg., Gynec. & Obst.* 66:810 (April) 1938.
11. Sharp, G. S., and Freeman, R. G., Cancer of the Lip. *Eye, Ear, Nose & Throat Monthly* 23:17 (January) 1944.

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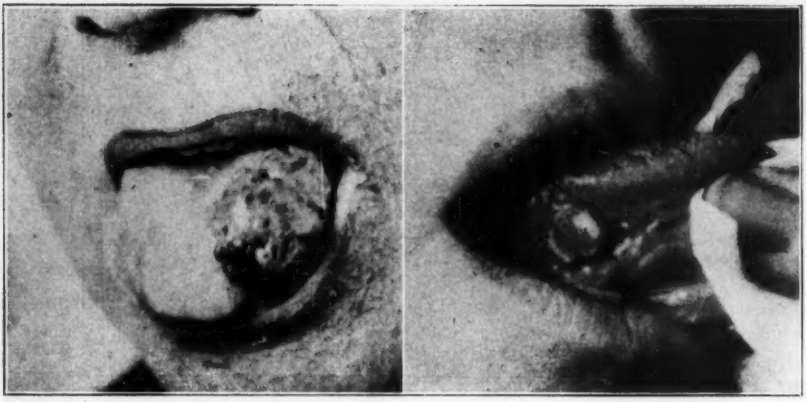

Fig. 4.—Left: Gumma. Right: Papilloma of tongue with superimposed leukoplakia

in almost all patients.[1] Yet, even as to mouth cancer in edentulous patients, there exists considerable divergence of opinion among competent authorities. A few years ago Figi,[12] reporting on experience gathered at the Mayo Clinic, concluded that cancerous growths involving the oral cavity "are infrequently encountered in edentulous persons and, when they do occur, are almost always of an inactive type." A recent statement by Martin,[1] on the other hand, strongly suggests that of patients at Memorial Hospital, New York City, who have oral cancer, a higher per cent are edentulous. But whatever the facts relating to this specific aspect of the problem may prove to be, the whole issue is clear enough to warrant at least tentative conclusions that (1) according to our present understanding of the cause of malignant degeneration, no one single factor causing chronic irritation or constant minor trauma can be held responsible for the ultimate development of a cancerous lesion, but that (2) injuries caused by various dental conditions are apt to start a sequence of changes within the oral tissue which sooner or later may terminate in a state of malignant degeneration.

Bridges and dentures are often the cause of mechanical injury to adjacent oral structures. In a recently reported series of cases of oral cancer, 31 per cent of the women patients were found to wear ill-fitting dentures.[13] It must, however, remain doubtful whether indeed all lesions of this type can be traced to irritation caused by pressure against the alveolar ridge or to rubbing against the mucous membranes of the palate, lip, cheek and gingiva. In certain instances the reverse may be true; a growth originating in tissues adjacent to dentures, but not in direct contact with them, may change the surface contour and thus cause the dentures to become ill-fitting.[1] But in any case, careful periodic check of dentures and bridges is required, as the contours of jaws and gums change, while dentures remain static and need readjustment.

Dentures, however, are not a potential source of mechanical irritation only. Chemical irritants have been encountered in cheaper dentures made of an inferior quality of material. Such substances are apt to contain arsenic, fluorine and various coal

12. Figi, F. A., Malignant Diseases of the Mouth. *J.A.D.A.* 26:1979 (December) 1939.

13. Beiswanger, R. H., and Stenstrom, K. W., Carcinoma of the Cheek, Alveolar Process, Floor of Mouth and Palate. *Radiology* 44:213 (March) 1945.

tar dyes, which may act upon a sensitive mucous membrane. Furthermore, rubber, used as a denture material in the past, becomes porous after a time, and microscopic fissures and interstices develop between the artificial teeth. These small apertures will harbor food particles, and as it is impossible to clean the minute crevices, decay, disagreeable odor and irritation will ensue. The possibility of damage after stimulation by an electrical current, created through the contact of metal fillings or prostheses of substantially different atomic weight, appears now more remote than had formerly been assumed.[3]

Physiologic Changes

The third group of precancerous lesions includes a number of physiologic changes within the oral mucosa. Although the various dental disorders contribute greatly to the development of such morbid states of the oral mucosa, these degenerative lesions nevertheless have to be considered independently. The changes in the mucous membrane of the different structures of the mouth are, in all probability, not strictly localized conditions but represent the circumscribed appearance of a general disease; any deeper understanding of this type of precancerous lesion presupposes, therefore, an appreciation of the interacting influences of local stimulation and constitutional background.[4]

The various precancerous conditions brought on by degenerative changes of the oral mucous membranes include: leukoplakia, subacute or chronic inflammation; verruca; vascular injection; atrophy or hyperthrophy of the lingual papillae; erosion of the epithelium; perlèche; cheilosis; cheilitis exfoliativa; keratosis; inflammatory hyperemia of the mucous membranes, especially of the tongue, cheeks or gingiva; gingivitis; Plummer-Vinson syndrome (sideropenic dysphagia); fibro-epithe-

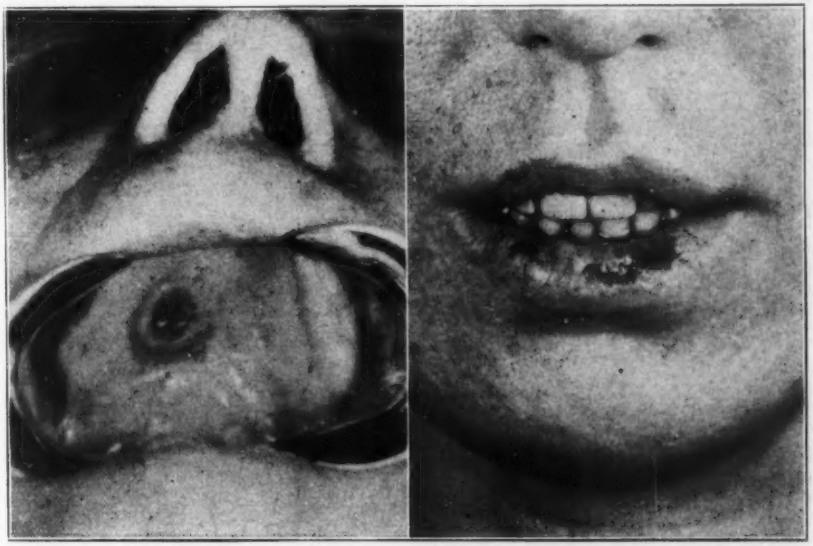

Fig. 5.—Left: Granuloma of palate. Right: Cheilitis of lower lip

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lial hyperplasia; erythroplasia of Queyrat, and the lesions of syphilis and tuberculosis. In addition to these relatively well defined disease entities, any gross localized tissue abnormality, erosion or ulceration which has resisted ordinary simple measures of treatment for over a month should be considered as precancerous.

Normally, the mucous membrane of the mouth is comparatively resistant to the development of cancer. But this normal resistance can be broken down through the development of a chronic state of tissue deterioration.

All degenerative lesions of the oral mucosa are hyperplastic in the sense that they are made up of more cells than are the tissues from which they originate.[14] Histologically, a series of changes can be noted: hypertrophy of the horny layer; fixation of the basal cell layer; hypertrophy of the epithelium; cell infiltration; increased vascularity, and edema.[15] These changes precede frank malignant degeneration.

In mucosa in this hyperplastic state there occurs distinct depression of cellular activity. The conclusion has been drawn that it is precisely in the face of such pathologic alteration of the cellular environment that formerly harmless carcinogenic factors may become tumor-producing, and that, consequently, the hyperplastic tissue cell turns neoplastic.[16] It is generally admitted that the malignant transformation occurs not throughout the hyperplastic lesion but in one or more very small groups of cells. Yet, it is impossible to identify in advance the cells in which this change will occur, nor can the time be ascertained beforehand.[14] In fact, development of even those growths which will later prove to be highly invasive is generally hesitant in the beginning. There appears to exist an early stage of primary neoplastic changes, before the tumor becomes aggressive through secondary neoplastic degeneration, superimposed upon the primary changes.[16] Evidence exists that the duration of this early stage varies in individual cases.[17] This factor is of the greatest importance, be-

14. Cowdry, E. V., Factors in Cancer Production. *Surg. Clin. North America* 24:985 (October) 1944.
15. Ashbury, H. H., Function of Dentist in Prevention and Care of Intra-Oral Cancer. *West Virginia M. J.* 34:1 (January) 1938.
16. Rous, P., Recent Advances in Cancer Research. *Bull. New York Acad. Med.* 23:65 (February) 1947.
17. Taylor, H. C., Jr., and Guyer, H. B., A Seven-Year History in Early Cervical Cancer. *Am. J. Obst. & Gynec.* 52:451 (September) 1946.

Fig. 6.—Left: Glossitis rhomboidea mediana. Right: Perlèche

Fig. 7.—Leukoplakia of the tongue

cause an early growth is not only small but constitutes intrinsically a less serious threat. In many instances, therefore, steps of immediate treatment can be taken to keep the malignant cells from dividing, thus checking further spread of the cancer.[16]

It was necessary to trace briefly the change from benign neoplastic precancer to the early stages of frank cancer. In a number of instances, histologic examination of tissue from a merely degenerated mucosal membrane shows an arrangement which closely simulates true neoplasm.[3] Yet, in many cases of prolonged irritation there occurs no evidence of canceration,[18] although at times malignant growths start in previously normal tissue within the oral cavity[19] only to become relentlessly aggressive within a few months after the first symptoms have been noted.[20] We may, therefore, summarize by stating that even though nothing can be stated definitely about the prognosis of a degenerative lesion in any individual instance, experience tells us that these conditions are highly dangerous in a great percentage of cases and, therefore, always demand immediate specific treatment.

Among the hyperplastic lesions of the oral mucosa, leukoplakia is not only more frequent than others but is also relatively well understood. This disease is by no means restricted to the oral cavity, including the lips, but is also encountered in the vagina, the bladder, and other mucous membranes of the body.

18. Logsdon, C. M., Malignancy of the Oral Cavity Region. *J.A.D.A.* 26:879 (June) 1929.

19. Simpson, F. E., and Breed, J. E., Cancer of the Mouth. *Radiol. Rev. & Mississippi Valley M. J.* 60:52 (March) 1938.

20. Gardham, A. J., Classification of Mouth Cancer in Relation to Treatment and Prognosis (Hunterian Lecture). *Lancet* 1:677 (March) 1939.

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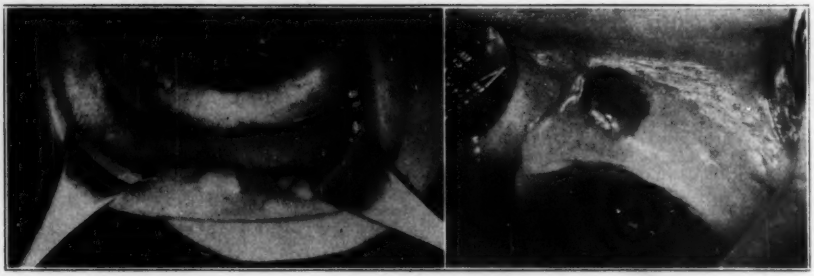

Fig. 8.—Left: Hypertrophic leukoplakia. Right: Giant-cell epulis

If this fact is taken into consideration it may be seen that an inherent tendency to hyperkeratosis may exist, which manifests itself subsequent to local irritation.[21]

Leukoplakia

Leukoplakia in its earlier stages appears as a nonpalpable, faintly translucent white discoloration. If the process is allowed to continue, the lesion turns opaque white, and a papillary growth of fine granular texture develops, which is easily palpable.[22] In a last stage, the mucous membrane becomes thick and stiff, and erosion and ulceration take place. It progresses very slowly, but once the ulcerative phase has been reached it responds poorly to treatment and the lesion must be considered as dangerous if, indeed, it has not already become malignant.[3] The pathologic process underlying the development of leukoplakia consists of an attempt of the mucous membrane to protect its sensitive deeper layers from chronic irritation through the development of circumscribed hypertrophy of the horny layer. At the same time the subcutaneous stratum is invaded by small, round cells, a process typical of chronic inflammation.[11] The white plaques, characteristic of the lesion, are cornified, dead cells heaped over abnormally thickened, irritated squamous epithelium.

Summary

This brief outline of the precancerous lesions and conditions appearing within the oral cavity can, of necessity, only touch on the multitude of problems involved. The dentist, in order to be able to fulfill an important part in the prevention of mouth cancer, which is his privilege and duty, must become increasingly familiar with the symptoms, causes, diagnosis, prognosis and therapeutic problems involved in each of the various conditions. What should be abundantly clear is the wide field open to the diagnostic ability of the dentist, a field which at the present time appears to be extending further as the intimate relation between degenerative changes within the oral mucosa and the condition of the rest of the alimentary canal is increasingly better understood.—*595 East Colorado Street.*

21. Eichenlaub, F. J., Leukoplakia Buccalis. *Arch. Dermat. & Syph.* 37:590 (April) 1938.
22. Eckert, C. T., and Petry, J. L., Carcinoma of the Lip. *Surg. Clin. North America* 24:1064 (October) 1944.

THE BLOOD AND ORAL SURGERY

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THE DAILY DEMANDS placed on the oral surgeon as diagnostician and therapist for oral diseases and injury preclude the necessity for familiarization with all possible intercurrent systemic diseases. The high degree of technical skill required in carrying out a surgical procedure can be attained with minimal scientific knowledge. The successful outcome of the procedure, however, depends largely on the application of an abundance of previously acquired facts. The surgeon is forced to keep well versed in the fundamentals of clinicopathology to enable him at all times to exercise adequate judgment in the establishment of a diagnosis, evaluate the patient's status, prepare the patient for surgery and care for him postoperatively.

The importance of diseases of the blood to those primarily interested in oral health has been recognized for a long time. Because of the seriousness of this group of diseases, and the fact that one is apt to become neglectful without constant contact with the problems involved, a review of basic fundamentals of present day knowledge of blood dyscrasias is justified.

Normal Blood

The knowledge of the normal composition of blood is a prerequisite to the understanding of the abnormal. The blood consists of a colorless circulating liquid plasma holding in suspension a number of cellular elements: erythrocytes, leukocytes and platelets. The circulating volume averages about 6,000 cc. and remains fairly constant despite variations in fluid intake.

The erythrocytes or red blood cells contain the hemoglobin necessary for the transportation of oxygen from pulmonary alveoli to tissue. They arise chiefly from the bone marrow in the adult and in their most immature form are nucleated and known as megaloblasts. As they mature and change morphologically they become erythroblasts, normoblasts and then lose their nuclei to become young adult cells, the reticulocytes, and finally the mature erythrocytes. Evidence of increased hematopoietic activity is apparent when the immature forms are seen in the peripheral blood.

One cubic millimeter of normal blood contains 4,500,000 to 5,500,000 erythrocytes. Their life span is approximately one month after which they are taken up by the reticulo-endothelial system and broken down chiefly into iron and bilirubin.

The average amount of hemoglobin held by the red blood cells is 15.6 Gm. per 100 cc. of blood. This figure usually is regarded as representing 100 per cent. The relation between the hemoglobin and the red blood cells gives the color index and is calculated by dividing the percentage of hemoglobin by the percentage of red blood cells. Five million red blood cells per cubic millimeter is regarded as being 100 per cent.

The leukocytes or white blood cells are primarily the phagocytes, assisting in the defense against infection. One cubic millimeter of normal blood contains

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5,000 to 9,500 of these cells of which there are three principal forms: the polymorphonuclear or granular leukocyte, the lymphocyte and the monocyte.

Of the polymorphonuclear group there are three types: the neutrophil, eosinophil and basophil. The neutrophils form about 60 per cent of the total number of leukocytes. The most immature form is known as the myeloblast and as it passes through successive stages to maturity it becomes the myelocyte, metamyelocyte and the stab form before becoming the neutrophil. An increase in the presence of the more immature forms often is spoken of as a "shift to the left" and is seen notably in infection. The eosinophils and the basophils differ from the neutrophils chiefly in their staining characteristics and vary in number in the presence of certain disease processes. The former constitute 2 to 5 per cent and the latter usually less than 1 per cent of the total white cell count.

The lymphocytes differ from the polymorphonuclear cells in morphology and parentage. These cells are lymphogenous in origin, arising in organs containing lymphoid tissue, and make up 20 to 30 per cent of the white cell count. The most immature form is known as the lymphoblast.

Approximately 5 per cent of the total leukocytes are monocytes. Their formation is in dispute. Immature forms are recognized in the form of the monoblast and the promonocyte before the monocyte normally is seen in the peripheral blood.

A normal routine complete blood count therefore may be summarized as follows:

Hemoglobin: 15.6 Gm. (100 per cent)
Red blood cells: 4.5 to 5.5 million
White blood cells: 5,000 to 9,500
  Polymorphonuclear (granulocyte) cells:
    Neutrophils: 60 to 70 per cent
      Nonfilamentous (young) cells: 9 per cent
    Eosinophils: 2 to 5 per cent
    Basophils: 0 to 1 per cent
Lymphocytes: 20 to 30 per cent
Monocytes: 5 to 9 per cent

Abnormal Blood

Disorders of the blood or blood forming organs result in abnormalities of the corpuscular composition or in the coagulating ability of the circulating blood. The fact that blood dyscrasias may have many manifestations and far reaching serious significance indicates that a complete blood count should be a routine diagnostic performance when systemic disease is suspected or extensive oral surgery contemplated.

I. Anemias

The term anemia applies to a deficiency in the concentration of hemoglobin or erythrocytes below the normal values. Anemia is not a disease in itself. It is a manifestation of disturbances of the mechanisms for the formation and disposal of red cells. The picture of anemia is seen when there is an increased removal of red cells from the circulation or if there is an impairment of blood formation. Increased removal of red cells, or blood loss, is the result of hemorrhage or hemolysis. Impaired formation results from (1) a deficiency of a specific sub-

stance required for blood formation; (2) a destruction of the erythropoietic cells of the bone marrow; (3) a displacement, or crowding out, of the erythropoietic cells, and (4) a depression of erythropoiesis.

Anemia of Acute Blood Loss.—The oral surgeon frequently is confronted by this anemia. The signs and symptoms manifested are characteristic. The patient feels weak, dizzy, faint and complains of thirst. He may be nauseated and vomit. The pulse is rapid and weak; the skin pale and cold; the respirations are rapid and gradually become shallow. The blood pressure gradually falls to a critical level and if blood loss continues it sharply falls as the picture of shock supervenes.

The earliest change in the blood picture of a person suffering from a sudden severe blood loss is a polymorphonuclear leukocytosis with an increase in the number of immature forms. This is seen within relatively few minutes after hemorrhage and increases throughout the first few hours. As the fluid content of the blood decreases the concentration of hemoglobin and red blood cells may not change. However, as fluid is withdrawn from the tissues to make up the deficit, the diminution in cells and hemoglobin becomes evident. This manifestation, along with the presence of immature red cells, is noted within two to three days.

The management of acute blood loss revolves around replacement. Whole blood transfusions from a compatible donor is the ideal treatment. Constitutional reactions can be alleviated temporarily by the administration of 5 to 10 per cent glucose by venoclysis.

The anemias resulting from an impairment in blood formation include an extensive list of disturbances, a detailed discussion of which is beyond the confines of this paper. A few notable forms, because of their frequency, are worthy of mention.

Lack of a specific substance required for blood formation results in a "deficiency anemia." The deficiency results in an interference with red cell development at a definite stage of maturation producing morphologic changes in the circulating red cell. The recognition and interpretation of these changes make detection and therapy of the specific deficiency possible. The deficiency may be in the form of the "liver principle," iron, protein, vitamins or hormones.

Deficiency in the "Liver Principle."—This erythrocyte maturing factor results in an anemia characterized by a large cell of normal hemoglobin concentration (macrocytic normochromic anemia) and by megaloblastic proliferation. The liver principle is composed of two factors: an intrinsic, derived from gastric secretion, and an extrinsic derived from food ingestion. Lack of the intrinsic factor produces a clinical picture described as pernicious anemia. A diet deficient principally in animal proteins and the vitamin B complex results in a macrocytic anemia but with clinical features unlike pernicious anemia.

Pernicious Anemia.—This anemia is characterized by a gradual onset of weakness, fatigue, anorexia, gastro-intestinal symptoms and neurologic disturbances. The disease usually appears in middle life and is more common in members of the Nordic race. The patient may complain of a progressive weakness, loss of appetite, abdominal discomfort, constipation or diarrhea and a sore tongue. There may be numbness and pricking sensation of the extremities, paralysis and muscular atrophy. Dyspnea, precordial distress, headaches and vertigo may be symptoms.

Examination of these patients discloses a lemon-yellow skin, a splenohepatomegaly, neurologic signs and, of importance to the dentist, a beefy red sore

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tongue with atrophy of the filiform and fungiform papillae. This gives to the dorsum of the tongue a characteristic bald smoothness.

Laboratory data reveals an anemia with the deficit in the number of erythrocytes (color index over 1), a leukopenia, a thrombocytopenia and an increase in blood bilirubin. The smear discloses abnormally large erythrocytes showing marked variation in size and shape (anisocytosis and poikylocytosis). Absence of gastric hydrochloric acid (achlorhydria) is a constant associated finding.

Treatment is confined to arresting the disease by replacement of the required principle and must be continued throughout life. While the blood abnormalities and most of the clinical features disappear under treatment, the severe neurologic signs and gastric achlorhydria persist.

Synthetic folic acid is of value in the treatment of the macrocytic anemia. It is possible that this substance is needed by the developing red cell to complete its development in the marrow before being released into the circulating blood.

Deficiency of Iron.—This deficiency is a frequently encountered cause for anemia. These are microcytic hypochromic (small cells with low hemoglobin) anemias and usually present a normoblastic reaction of the marrow. An average 150 pound man has approximately 4.0 Gm. of iron in his body. Three grams of this are present in hemoglobin; 0.5 Gm. are contained within tissue cells, and 0.5 Gm. are stored for reserve in the liver.

Approximately 100 mg. of iron are required daily for the replacement of red bloods removed from the circulatory system. Although the conservation of iron from the breakdown of hemoglobin is remarkably efficient, some of the metal is lost through excretion and requires replacement by ingested foods. The average daily diet is estimated to contain less than 16 mg. of iron of which about one-half is available for absorption. During such physiologic processes as growth, pregnancy and menstruation a deficit is apt to occur unless compensated by increased intake. Iron deficiency usually is manifested in pathologic processes such as chronic blood loss, impaired iron absorption, hemolysis and low iron intake.

Microcytic hypochromic anemia accompanies many disturbances of the body. It is a fairly frequent finding in women between the ages of 30 and 50 and often is associated with a menorrhagia.

Aplastic Anemia.—This disease belongs under the group of anemias resulting from impairment of blood formation but specifically is the result of a destruction of erythropoietic cells. The erythropoietic tissue characteristically is replaced by fat. The causative agent may be known and often is attributed to chemicals, such as arsenicals, benzol or gold, or excessive exposure to radiation.

The disease is progressive and is manifested by a decrease in all marrow elements: red cells, white cells and platelets. Although the quantity of red cells is diminished, the quality remains unchanged. Evidence of erythropoietic activity cannot be detected by an examination of the peripheral blood.

The symptomatology is identical with that of any other severe anemia except that spontaneous hemorrhage is likely to occur.

Myelophthisic Anemia.—This disease is similar in many respects to aplastic anemia except that loss of erythropoietic cells is caused by their displacement with an invasion of the marrow by neoplastic tissue. Because of the pressure exerted on the developing cells by the invading tissue, regions of erythropoiesis may be released from the medullary spaces. Therefore, immature red cells are seen in increased numbers in the circulating blood.

The oral surgeon frequently is confronted with the problem of anemia. The

problem may be one of diagnosis. The unusual pallor of the oral mucosa and the presence of glossitis may offer a diagnostic challenge. Hypersensitivity and tenderness of the tongue may be an early symptom of pernicious anemia. From this the typical raw, smooth or bald tongue may develop. The changes are more apt to be marked on the lateral borders and tip. Occasionally vesicles and ulcers will appear as a result of the inflammation.

Changes of the tongue are not confined necessarily to the clinical picture of pernicious anemia. They also are seen in other macrocytic and microcytic hypochromic anemias, gastric neoplasms, cirrhosis of liver, intestinal obstructions and after gastrectomies. It is interesting to note that this finding is characteristic of the deficiency anemias. Experimental data has indicated that the deficiency is produced by the failure of some functions of the stomach. The deficient substance producing the tongue changes probably belongs to the vitamin B complex group. It is unusual to note tongue findings in any of the anemias outside of the deficiency group.

Aplastic anemia frequently produces spontaneous intra-oral hemorrhage, and oral petechiae are not an uncommon finding.

The anemic patient presenting himself for oral surgical procedures demands special consideration. Regardless of the type of anemia, the initiating factor must be identified so that proper treatment can be instituted. This demands cooperation with the internist because correction of an intercurrent systemic disease is outside the realm of oral surgery.

It is obvious that anemias must be controlled carefully, with close observation of symptoms and laboratory findings, before and during the surgical procedure. Extreme care must be exercised in handling patients with pernicious anemia as they are easily disturbed by fright and anxiety such as that produced by a surgical procedure. Under such stimuli an exacerbation of the disease may occur and the blood count may drop suddenly.

Submitting a person with uncontrolled anemia to extensive oral surgery may have serious effects. Because the anemic patient has diminished oxygen-carrying capacity of the blood he is highly susceptible to shock. Further, there is decreased resistance with a decided predisposition toward postoperative infection. Tissue repair may be delayed and inadequate. Anemias accompanied by a thrombocytopenia also present an impaired clotting mechanism, and prolonged hemorrhage may follow even minor surgery. Blood loss must be avoided in the treatment of anemic patients regardless of their status.

Except for emergency operations, no surgery ever should be undertaken on patients presenting a hemoglobin level below 60 per cent or an erythrocyte count below 3,500,000 per cubic millimeter. Postoperative correction of an anemic state should cover as long a period as possible to enable the patient to replace blood loss and adequately to repair tissue.

II. Polycythemia

Any condition associated with an erythrocyte count above normal limits is regarded as polycythemia. Primary polycythemia, however, might be differentiated from the secondary form. The latter is a compensatory rise in the red cell count to meet a changed respiratory or circulatory situation.

Polycythemia is a chronic, slowly progressive, eventually fatal disease characterized by excessive erythroblastic activity and probably represents a form of

neoplasm. The total volume of the blood is increased; the increase is due to a greater red cell mass. Erythrocyte counts usually vary between 7 and 11 million with hemoglobin ranging between 20 and 40 per cent above normal.

Because the oxygen requirements of the patient are unchanged, the venous blood is abnormally rich in oxygen and the skin takes on a red cyanotic appearance. There are often accompanying lassitude, vertigo, tinnitus, weight loss, dyspnea and gastro-intestinal symptoms. Distention of the spleen by the surplus cells usually causes an enlargement of that organ.

The increased viscosity of the blood and distention and engorgement of the capillary vessels may produce thrombosis and spontaneous hemorrhages. Surgical measures attempted on these patients may result in postoperative hemorrhage that is most difficult to control.

Treatment is confined to repeated venesections, irradiation and the use of drugs having the ability to destroy red blood cells. The use of radioactive isotopes recently has been employed with gratifying results. This treatment is most satisfactory when combined with repeated venesections. However, life expectancy has not been increased greatly, and relatively few patients live longer than ten years after the establishment of the diagnosis.

III. Agranulocytosis

Agranulocytosis is an acute fulminating disease of sudden onset characterized by fever and prostration and accompanied by sore throat and necrotic ulcerations of the mucous membranes. The disease may be ushered in by a feeling of weakness and malaise; high fever, chills, nausea and marked prostration follow rapidly.

The peripheral blood characteristically shows extreme leukopenia and neutropenia. The total white cell count may go below 500 cells per cubic millimeter with 10 per cent or less neutrophils. At the height of the disease it is not unusual to find that the lymphocytes make up 98 per cent of the differential count.

If not treated, patients with the acute form of the disease may die within a week. The subacute and chronic forms may persist for years. Marked susceptibility to infections of the skin and mucous membrane is a manifestation of the disease.

The literature has been filled with descriptions of the causative factors of this disease, with particular emphasis on the role of aminopyrine and related compounds. Other notable offenders have been the sulfonamide derivatives, gold, benzol compounds and dinitrophenol. An occasional report of penicillin as a precipitating factor may be found in recent literature. It is agreed generally that an allergic factor must be present for the production of the disease. The amount of the drug consumed evidently is not important, as fatal agranulocytosis may follow even minimal doses; therefore all incriminated drugs must be used with the greatest caution.

There is no specific therapy. Obviously all drugs suspected of possibly initiating the disease must be withdrawn immediately. Favorable results have been reported with the use of blood transfusions, Pentnucleotide (a leukocyte stimulant produced by Smith, Kline & French Laboratories) and yellow bone marrow. A general supportive program and local and general measures for the control of the infection must be instituted.

In view of the fact that the earliest symptoms of agranulocytosis frequently arise in the mouth, and because this disease often follows the use of drugs em-

ployed in oral surgery, the disease is of particular importance to the oral surgeon. The early diagnosis of agranulocytosis may be an all important factor in saving the life of the patient. It has been stated that symptoms of agranulocytosis appear when the number of polymorphonuclear neutrophils drops below 800 cells per cubic millimeter. It has been supposed that the necrotic lesions occur first on the oral mucosa because of the constant trauma the oral mucosa is subjected to in the presence of the large number of micro-organisms. While these micro-organisms are ineffective with normal leukocytic defense, they may cause severe secondary infection when the defense is removed. Oral surgical procedures carried out in the face of agranulocytosis obviously would invite disaster.

IV. Infectious Mononucleosis

Infectious mononucleosis is an acute infectious disease occurring most frequently in young adults. The disease is of long duration with recurrent exacerbations and prolonged convalescence. There is usually fever, lymphadenopathy and a tendency toward secondary upper respiratory infections. Severe sore throat and tender cervical lymph nodes are common findings.

The initial stage of the disease is characterized by a leukopenia and neutropenia; after a few days the white cells increase in number with the presence of atypical immature lymphocytes that may reach 80 per cent of a total count of 20,000.

The diagnosis is dependent on the clinical picture and characteristic blood picture. The Paul-Bunnell agglutination test is of definite value. The illness is confused most often with acute leukemia; however, the prognosis of infectious mononucleosis is very favorable, although the patient may have weakness and fatigue for a long period following its acute course.

The oral surgeon must keep in mind that a persistent sore throat and cervical lymphadenopathy is suggestive of this disease.

V. Leukemias

Leukemia is probably a neoplastic disease of the leukopoietic tissue. Its chief difference from other neoplasms is that its origin is diffuse rather than from a solitary primary site. The tissues involved are primarily the spleen, lymph nodes and bone marrow. The symptomatology, although bizarre, usually includes progressive fatigue and weight loss, fever, abdominal discomfort with gastrointestinal symptoms, pruritis, spontaneous hemorrhages and occasional pain over the long bones.

Physical examination discloses evidence of weight loss, pallor, a generalized discrete enlargement of lymph nodes and a splenohepatomegaly. There are often severe oral lesions, in the form of necrotic ulcers and gingival hyperplasia, that may be among the earliest symptoms of the disease.

Three principal forms of leukemia are recognized: myeloid, lymphatic and monocytic. The disease may be acute or chronic, but it is always progressively fatal.

Myeloid (Myelogenous) Leukemia.—The chronic form of this disease is the commonest of leukemias. There is, as in the other forms of leukemia, a marked leukocytosis often up to and sometimes exceeding 600,000 per cubic millimeter. All stages of polymorphonuclear or granulocytic development are represented in the marrow and usually in the peripheral blood. The usual duration of life

after diagnosis is about three years. The disease may terminate in the acute form, from progressive cachexia or from secondary complications.

Acute Lymphatic Leukemia.—This disease is the commonest of the leukemias of early childhood, while the chronic form strikes principally in elderly persons. Blood examination discloses a predominance of lymphoblasts with 90 per cent or more of the white cells being of the lymphocyte group. The course of the chronic type is likely to be of a longer duration than the myeloid form—five years being the average duration.

Monocytic Leukemia.—Although this is the least common of the leukemias, the incidence appears to be on the rise. The total white count rarely exceeds 50,000 with the monoblasts and promonocytes predominating. Oral symptoms (sepsis and ulcerative stomatitis) are found most frequently in this form. The duration is variable. The patient may succumb within a few weeks after onset, or the disease may last for years.

The displacement of red cell and platelet forming tissues of the bone marrow seen in the majority of leukemias results in anemia and thrombocytopenia. Therefore a spontaneous hemorrhagic tendency is commonly present, and bleeding into the skin from the mucous membranes is not an uncommon finding. The first evidence of the disease may be profuse hemorrhage following the removal of a tooth or minor traumata.

Roentgen irradiation long has been the best available treatment for leukemia. The prognosis, however, is unchanged, although the duration of life may be prolonged a few months. Radioactive isotopes have been used in recent years to replace roentgen irradiation chiefly because the radiation sickness seen in the latter is not encountered.

Certain chemicals, urethane and nitrogen mustard, have an effect similar to roentgen irradiation. They first were employed therapeutically in leukemias because they were found to produce a leukopenia. Definite evidence as to curative effect is not obtainable because both drugs are still in the experimental stage.

The significant oral symptoms, spontaneous bleeding, necrotic ulcerations and marked gingival edema and hyperplasia, are usually severe at the terminal phase of the chronic leukemias and at the initial stages of acute forms.

The removal of teeth for a patient afflicted with acute leukemia may be followed quickly by death. The widespread necrosis that results from such a procedure produces overwhelming toxicity.

Urgent dental extractions for patients having chronic leukemia may be carried out during a remission of the disease after all necessary precautions have been taken. The patient should be hospitalized and placed under competent medical supervision. Careful blood examinations to determine the degree of anemia, number of white cells and platelets along with clotting and bleeding time is imperative. Pre- and postoperative transfusions may be necessary. Uncontrollable hemorrhage, severe infection and the possibility of a sudden exacerbation of the disease always must be anticipated. Close relatives should be informed of these possibilities.

Hemorrhagic Tendencies

The blood possesses the property of coagulation to prevent significant blood loss should disruption of the integrity of a vessel wall occur. A blood clot consists of a fibrin network that contains the corpuscles, which contracts and ex-

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presses a serum, sealing the break in the vessel wall. The clotting of blood depends on the transformation of fibrinogen in the circulating blood to the insoluble fibrin of the clot. This has been shown to be caused by the action of thrombin on the fibrinogen. Thrombin, in turn, exists in the blood in the form of prothrombin which must be acted on by calcium ions before it can be converted to thrombin. Besides calcium, another factor is necessary. This factor is furnished by tissue juice or blood platelets and has been designated by the name "thromboplastin." It generally is agreed that in the conversion of inactive prothrombin to active thrombin, both the thromboplastic factor and calcium ions are concerned, but the precise nature of the mechanism is a matter of theory.

The abnormal tendency to bleed is a manifestation of a defect in this clotting mechanism, including inability of the clot to contract; or deficiencies in capillary permeability.

The blood platelets (thrombocytes) are essential to the clotting mechanism. There are about 250,000 per cubic millimeter and they are 2 to 3 μ in diameter. Their disintegration results in the release of thromboplastin. Their life represents only a few days after which they are removed by the reticulo-endothelial system, principally the spleen. An increased number of platelets (thrombocytosis) is associated with a tendency toward thrombosis, while thrombocytopenia results in hemorrhage.

VI. Purpura

Purpura is a symptom complex manifested by spontaneous hemorrhages. There are two major types: thrombocytopenic and nonthrombocytopenic. The thrombocytopenic purpura is classed further in groups called primary (essential) and secondary (symptomatic).

Primary Thrombocytopenic Purpura.—The etiology is unknown, and it occurs most frequently in young persons. The chief clinical feature is bleeding from the mucous membranes and subcutaneous hemorrhages. The first indication of the disease may be uncontrollable hemorrhage after the removal of a tooth. Petechiae, conjunctive or generalized, may appear spontaneously beneath the oral mucosa, and marked ecchymosis may follow minimal injury.

The laboratory findings are decisive. There is a marked decrease in the number of blood platelets; the bleeding time is prolonged greatly while clotting time remains normal; clot retraction is delayed, and the tourniquet (Rumpel-Leede) test indicating degree of capillary resistance is positive.

A splenectomy usually brings about complete recovery. Most surgeons report remarkable immediate control of bleeding following ligation of the splenic vessels.

Secondary Thrombocytopenia.—The secondary form occurs as a sequence to some other condition, such as infections, toxic reactions to chemicals or physical conditions, allergies, roentgenotherapy or in conjunction with a blood dyscrasia.

Elective surgery in any form, of course, definitely is contraindicated in patients with thrombocytopenia. Emergency surgery may be done following preparation by multiple transfusions of compatible blood. Improvement persists only as long as platelets are in the circulating blood in sufficient number to permit the clotting mechanism to occur.

Nonthrombocytopenic Purpura.—This pertains to purpuric manifestations without decrease in the number of platelets. It occurs in association with various

diseases—notably allergies, infections, nutritional deficiencies, intoxications and in cachectic and debilitated persons.

The differential diagnosis between thrombocytopenic and nonthrombocytopenic purpura is based on laboratory and clinical data. Platelet deficiency and prolonged bleeding and clot retraction time seen in the former are not evidenced in the latter.

VII. Hemophilia

Hemophilia, a constitutional disorder, is sex linked in transmission. The disease is seen only in males but is transmitted by females. The increased bleeding tendency is so severe that few hemophiliacs survive infancy. Bleeding that occurs from the slightest injury may continue to exsanguination and death. Spontaneous hemorrhages do not occur.

The only significant laboratory finding is a delayed clotting time. The bleeding time is normal. The mechanism of the delayed clotting depends on resistance of the platelets to disintegration and, therefore, failure to release thromboplastin.

Surgical procedures, however minimal, on such a patient without preoperative recognition of the disease often will result in death from uncontrollable hemorrhage. No specific treatment is possible, but temporary control can be obtained by repeated blood transfusions. The removal of teeth must be preceded and followed by frequent blood transfusions. Of course, elective operations must be discouraged completely.

VIII. Prothrombin Deficiencies

Prothrombin, the precursor of thrombin and essential in the clotting of blood, is synthesized in the liver in the presence of a dietary factor named vitamin K. The latter substance, found in nature in green leaves, is fat soluble; absorption from the intestinal tract requires bile. The exclusion of bile prevents the absorption of vitamin K and, consequently, causes a decrease in the synthesis of prothrombin.

Obstructive Jaundice.—This disease is accompanied frequently by a bleeding tendency. This tendency is due to a deficiency of prothrombin; the same hemorrhagic predisposition is found in any disease with manifestations of reduced fat absorption and consequently reduced vitamin K absorption. The blood normally contains such an excess of prothrombin that bleeding tendency on a prothrombin deficiency basis usually does not become apparent until the concentration of this substance has decreased to within 20 per cent of the normal value.

There are other disturbances with associated bleeding tendency on a basis of prothrombin deficiency. Disturbances of the intestinal absorptive surface, advanced liver disease and hemorrhagic disease of the newborn are in this group.

The significant laboratory findings are prolongation of the bleeding, clotting and clot retraction times. Of course, these findings are not diagnostic as they may occur in association with other blood dyscrasias.

Surgical procedures on patients having prothrombin deficiency, whatever the cause, will be complicated by severe postoperative hemorrhage. The initiating factor must be corrected. However the administration of one of the vitamin K preparations temporarily will restore a hypoprothrombinemia.

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IX. Scurvy

Although scurvy, or avitaminosis C, is a nutritional and not a blood disease, the associated hemorrhagic tendency makes mention of it here necessary. The disease usually manifests a marked gingival edema with tendency to spontaneous gingival bleeding. In addition there may be spontaneous hemorrhages into the skeletal muscles with occasional petechial hemorrhages into the skin.

The hemorrhagic tendency is based on an abnormal capillary fragility resulting from inadequate intracellular collagenous material. Presence of the avitaminosis certainly may be a factor in the production of prolonged postoperative bleeding.

Data acquired from routine blood examinations usually is not diagnostic, although there may be a platelet deficiency. Cevitamic acid blood level determination usually will reveal a level below 0.3 mg. per 100 cc.

A relatively simple test can be utilized as an office procedure to determine capillary resistance. An area 6 cm. in diameter is marked off in each antecubital fossa and a blood pressure cuff is applied about each upper arm and inflated to 35 mm. of mercury for fifteen minutes. With a 5-power hand lens the petechiae are counted. After one hour the procedure is repeated but the cuff is inflated to 50 mm. of mercury. The petechiae in the first stage are multipled by 2, and the petechiae count in the second stage is added to this; the result is designated as the "petechial index." A petechial index over 9 indicates increased capillary fragility.

A product known as rutin, developed by the United States Department of Agriculture, is thought to decrease capillary fragility in a high percentage of cases. It is recommended that dosage be 20 to 60 mg., three to four times daily for several weeks or until the petechial index becomes normal. Pharmaceutical houses have combined rutin with ascorbic acid on the rationale that both are indicated in the treatment of capillary fragility. Rutin, however, is still under investigation and its efficacy has yet to be confirmed.

Summary and Conclusion

It is difficult to present in detail the numerous links between the practice of oral surgery and hematology. The obvious fully developed oral manifestations of a blood dyscrasia are usually no problem to the oral surgeon. They are recognized immediately as being medical problems, and consultation with the internist is sought. The real pitfalls and hazards are those cases of blood dyscrasia in which the oral complication is relatively mild or may simulate oral disease on a purely local basis. Almost all of the diseases of the blood may exhibit oral signs, and it is important that the oral surgeon be aware constantly of these possibilities.

Examinations of the blood definitely must be requested whenever any of the following signs or symptoms are encountered:

1. Persistent oral infection which does not respond readily to local measures, or the presence of necrotic lesions of the oral mucosa.
2. Hemorrhage, either in the history, as a postoperative complication, as spontaneous bleeding from the gingiva or the presence of petechiae or unusual ecchymosis following minimal traumata.
3. Atrophy of the papillae of the tongue or persistent glossitis.
4. A persistent sore throat with cervical lymphadenopathy.

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An indication of a blood dyscrasia usually can be obtained by taking a routine complete blood count and where indicated a platelet count, bleeding and clotting time and capillary resistance test. Abnormal findings necessitate further investigation which has no place in the field of oral surgery. These simple laboratory determinations are relatively inexpensive, require only a short time and actually may be life saving measures.—*Department of Oral Surgery, School of Dentistry, University of Michigan.*

LIQUID DIET FOR INTERVAL FEEDING

Oral surgeons always must keep in mind that ordering a diet which is adequate in every particular does not insure proper nutrition, for it is common knowledge that hospital patients all too frequently send back on their trays a considerable portion of their food. Constant supervision, time consuming patience and kindly urging is often necessary to establish a proper level of consumption. These precautions may be difficult to accomplish in average hospital routines. To meet this problem, Dr. R. L. Varco and the nutritional laboratory of the University of Minnesota General Hospital have designed a supplementary liquid diet for interval feeding to insure an adequate intake of protein. This is an in-between diet—not brought and taken away with the trays.

Gastric II, or University II, or Varco II

(May be given by tube feeding or may be given by mouth)

Analysis of one day's portion—1,500 cc.

Carbohydrates	408.8 Gm.	Total calories	2,428
Protein	120.4 Gm.		
Fat	37.2 Gm.	Calories per cubic centimeter	1.63

Ingredients	Amount	Carbohydrate	Protein	Fat
Whole eggs	6	....	36.0	36.0
Egg whites	2	....	8.0	....
Skim milk powder	4 oz.	58.8	40.4	1.2
*Lactose or sucrose	300 Gm.	300	....	....
Skim milk	1000 Gm.	50	36.0	....
Salt	5 Gm.			
Total		408.8	120.4	37.2

*Beet or cane sugar may be substituted.

FALL MEETING OF ANESTHESIA SOCIETY

The American Society for the Advancement of General Anesthesia in Dentistry will hold a fall meeting on October 25, 1948, at Midston House, Thirty-eighth Street and Madison Avenue, New York. Dinner will be served before the meeting at 7 p.m.; the scientific session will begin at 8:30.—*M. Hillel Feldman, executive secretary, 730 Fifth Avenue, New York.*

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ROENTGENOGRAPHIC STUDY OF THE MANDIBULAR JOINT

A. A. Sulke, D.D.S.,
Jerusalem

As IN ALL fractures, the roentgenogram is a deciding factor in diagnosis of fractures of the mandibular joint, especially for distinguishing the displacement of the fragments, and is indispensable in examination and treatment.

It is advantageous to take the picture of the joint in two directions, lateral and anteroposterior or postero-anterior. Interpretation of pictures of the condyloid region is highly difficult, owing to the superimposition of the shadows of many bones. The superimposed shadows in the lateral direction are those of the zygomatic bone and sphenoid bone, vertebrae and atlas. Fry, Shepherd, McLeod and Parfitt[1] give examples of roentgenograms of real fractures and seeming fractures.

There are many methods for positioning and projecting in a lateral direction. I will mention the methods that have been found useful in cases of true and suspected fracture of the condyle.

Thirty Degree Lateral Position

The head and plate are declined 30 degrees from the horizontal plane, and the tube is in the vertical direction. In this position the sound joint near the tube is not superimposed on the injured side (Fig. 1, left).

Fifteen to Twenty Degree Angular Direction from the Vertical

The technic of this exposure is described by Fry and his co-workers.[1] A line is drawn from the external meatus to the outer canthus of the eye. One and a half

1. Fry, W. K., Shepherd, P. R., McLeod, A. C., and Parfitt, G. J., *Supplement to the Dental Treatment of Maxillo-Facial Injuries.* Philadelphia: J. B. Lippincott, 1943.

Fig. 1.—Left: Exposure made from lateral position at 30 degree angle. Right: Exposure from angular direction in near projection

centimeters from the meatus a second line is drawn upward at right angles. One inch along this line is the position of the central ray. The patient's head is placed in the true lateral position, and the tube is manipulated until the central ray passes through the determined spot and the opposite condyle head. The tube should not be more than 20 degrees from the vertical (Fig. 1, right).

Lateral Near Projection

The patient's head is placed in the true lateral position, the injured side on the plate, and the tube is set directly over or only 1 cm. above the sound joint (Fig. 2).

It is possible to make a near projection picture also in the position described under the preceding section. In this position the sound joint and the other bones are well separated in projection, and the injured joint near the film can be seen with almost no superimposition.

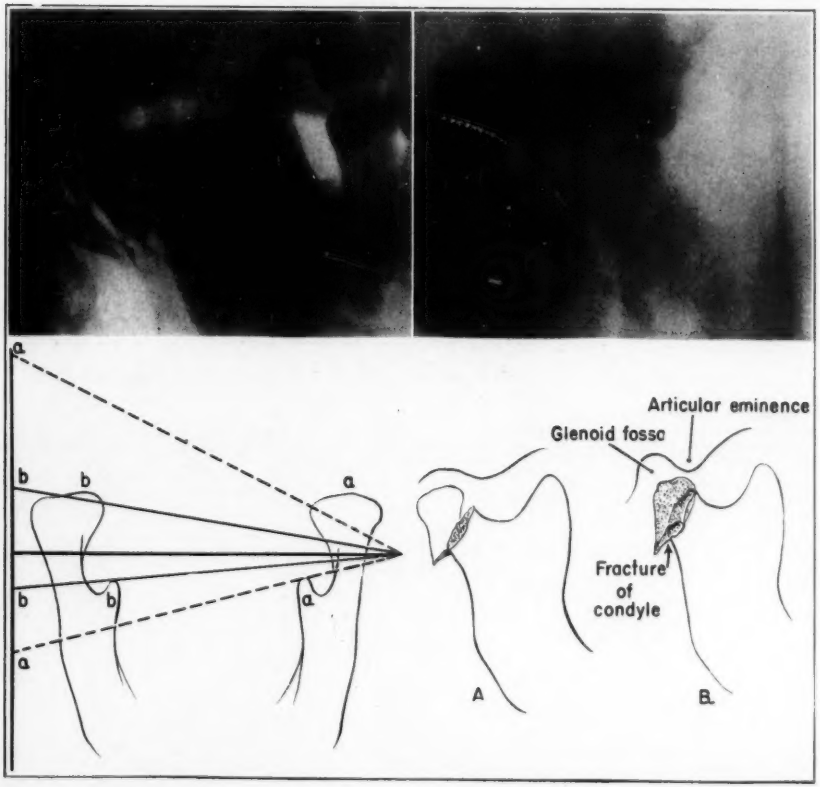

Fig. 2.—Left: Appearance of joint by lateral near projection. Sound joint (a) near tube is distracted. Injured joint (b) gives a clear picture. Right: Appearance of fractured condyle by lateral near projection and in tomogram

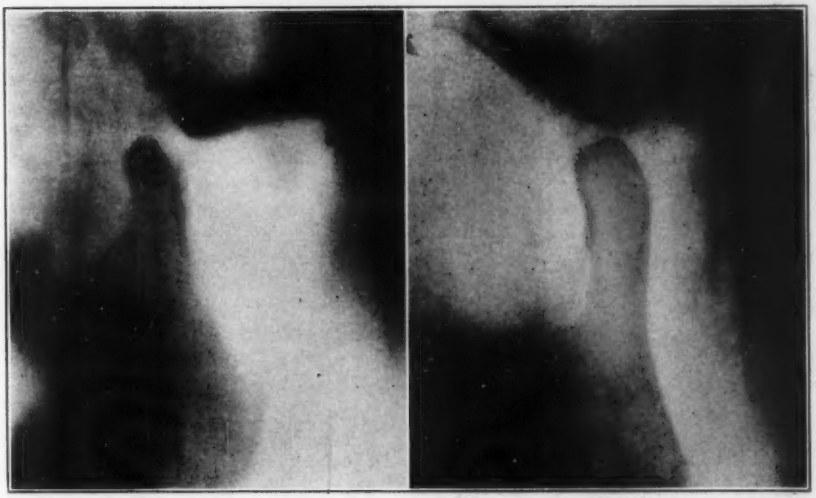

Fig. 3.—Left: Tomogram of condyle in luxation. Right: Tomogram of normal condyle

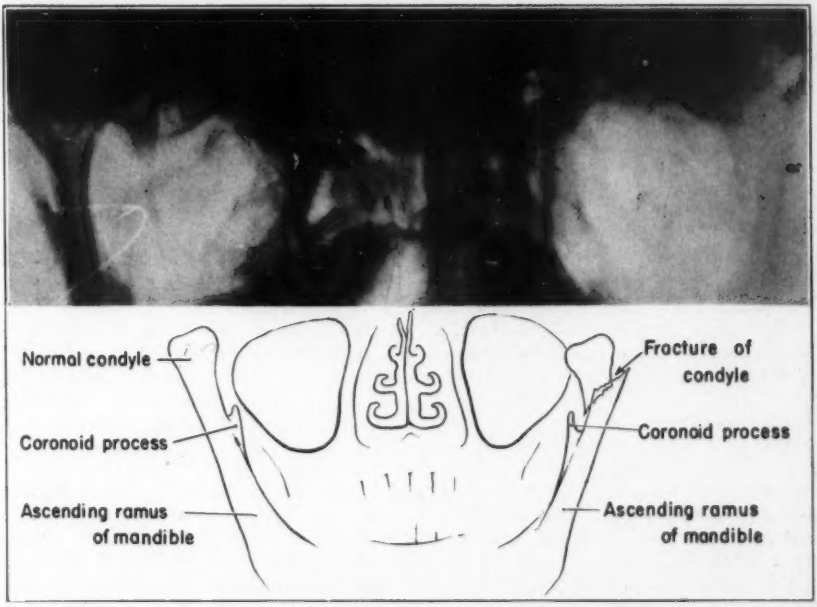

Fig. 4.—Anteroposterior projection of sound right condyle and broken left condyle

Tomographic Picture

The tomogram is taken with the x-ray tube circling 30 degrees 30 cm. above the injured joint which is placed in the center of the circle along which the tube is moving. By this method all structures which are hit by the moving rays are effaced and only the joint lying in the center of the circle gives a clear picture (Fig. 3).

The tomogram does not give distinct views of bony structure, but it discloses features which in ordinary roentgenograms are obscured by overlying and underlying bones. The fracture of the condyle and the superimposition of the displaced head over the mandibular collum are clearly visible to a degree that cannot be accomplished by the usual roentgenogram. By means of a tomogram the luxation of the condyle can be shown, from the point at which the condyle proceeds from the glenoid fossa to some distance before the articular eminence. In this picture the clearly outlined form of an uninjured condyle may be seen and compared with the shape of the fractured condyle shown in Figure 2, right.

This method is as yet complicated, but simplification will make the tomographic picture a very helpful measure.

Antero-posterior and Postero-anterior Projections

Usually the picture is taken with the face of the patient placed on the plate (Fig. 4), the tube vertical above the occiput. The shadows superimposed on the condyloid region are those of the sphenoid bone and the mastoid process, in that order. By chance, I found the anteroposterior direction to be the better. In a case of commotio cerebri the patient could not be turned without vomiting, even several days after the accident, and it was necessary to take the roentgenogram while he was lying on his back. The picture from this position is obviously clearer because the condyle region is nearer to the film. The occiput is laid on the plate and the tube is vertical above the nose. I recommend this method for the roentgenogram in all cases.—*Ben Jehudah Road.*

Classification of Maxillofacial Injuries. Lt. Colonel Daniel Klein, in *Plast. & Reconstr. Surg.* 2:133 (March) 1947.

Summary

Patients with maxillofacial injuries can be divided into three clinical groups. In the first classification are those patients exhibiting injuries to the face and neck of varying severity but without hemorrhage or obstruction of the airways. These patients are usually quite comfortable, need little sedation and show few, if any, signs of clinical shock despite the severity of the injuries. They safely tolerate extensive primary repair of the damaged parts. In the second classification are the same type of cases but suffering from noticeable loss of blood, respiratory obstruction or both. These patients are acutely ill and present many of the manifestations of clinical shock. Treatment is directed at the relief of this latter state by the replacement of lost blood and the establishment of an adequate airway. When these criteria have been met, primary reparative surgery of any degree may be carried out, as in the first group. In the third classification are the facial injuries with concomitant cerebral, thoracic, abdominal or extremity injury. Facial surgery will not compromise the treatment of these cases and should be done concomitantly for the end results of minimal scarring and deformity. Any apparently primary maxillofacial case exhibiting restlessness, drowsiness, altered blood pressure or pulse or respiratory rate not accounted for by hemorrhage or respiratory obstruction must be examined for a concealed injury.

SCHULLER-CHRISTIAN'S DISEASE WITH REPORTS OF CASES

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XANTHOMATOSIS of bone, Schuller-Christian's disease, Letterer-Siwe's disease and, more recently, solitary granuloma, lipogranuloma and eosinophilic granuloma of bone, are all names given by various authorities to a rare disease of the skeletal bones caused either by a disturbance of lipoid metabolism or some infectious agent.

There are two schools of thought on the cause of this group of diseases. Such men as Thannhauser,[1] Rowland,[2] Sosman[3] and Thoma[4] believe that they are caused by disturbances of lipoid metabolism and are characterized by deposits of lipoids in the bone marrow of any of the skeletal bones, but most often the flat bones and bones of the skull. Other authorities, Jaffe and Lichtenstein[5] and Farber,[6] argue that the lesions within the affected bones apparently represent a peculiar inflammatory reaction to some as yet unknown infectious agent and are characterized cytologically at the start by the presence of large numbers of histiocytes. In such an event, Schuller-Christian's disease must be removed from the category of primary disorders of the lipoid metabolism.

Character of the Disease

It has been the author's privilege, over the past twelve years, to observe 3 cases of Schuller-Christian's disease with approximately forty different lesions in almost all of the skeletal bones. Cultures taken from many of these lesions, urine analyses and blood studies of the patients have failed to show any positive agents of infection. Some cultures have shown Streptococcus hemolyticus, others yielded Staphylococcus aureus as the predominating organism and many cultures failed to show any growth. So it is difficult to understand how the later group of workers came to the conclusion that these lesions are caused by some infectious agent. They admit that such an agent is unknown as yet.

From the clinical observation, roentgenographic study and surgical findings, these lesions have much more of the character of neoplasms than regions of infected tissue. Therefore, I am inclined to favor the men who think that xanthomatosis is a disease of lipoid metabolism in which storage tumors of lipoid appear in various organs and bones.

For the present time it is preferable to the author to continue to speak of these lesions of bone as Schuller-Christian's disease. Solitary granuloma and eosinophilic granuloma of bone do not begin to describe the lesions which so closely resemble malignant neoplasms. It is true that, on pathologic study, specimens invariably appear as chronic inflammatory tissue with marked eosinophilic reactions; but from

1. Thannhauser, S. J., and Magendantz, H., Different Clinical Groups of Xanthomatous Diseases; Clinical Physiological Study of 22 Cases. *Ann. Int. Med.* 11:1662 (March) 1938.
2. Rowland, R. S., Xanthomatosis and the Reticulo-Endothelial System. *Arch. Int. Med.* 42:611 (November) 1938.
3. Sosman, M. C., Xanthomatosis (Schuller-Christian's Disease; Lipoid Histiocytosis). *J.A.D.A.* 98:110 (January 9) 1932.
4. Thoma, K. H., *Oral Pathology*. St. Louis: C. V. Mosby Co., 1941.
5. Jaffe, H. L., and Lichtenstein, Louis, Fibrous Dysplasia of Bone. *Arch. Path.* 37:99 (February) 1944.
6. Farber, Sidney, The Nature of "Solitary Eosinophilic Granuloma" of Bone. *Am. J. Path.* 17:625 (July) 1941.

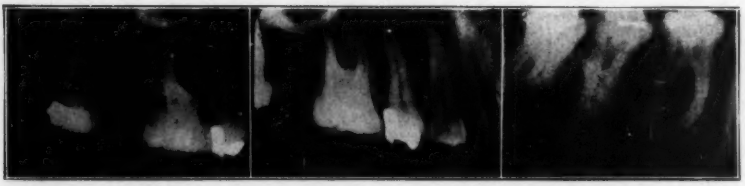

Fig. 1, Case 1.—Left and center: Roentgenographic evidence (April 29, 1937) for preoperative diagnosis of a radicular cyst involving the second bicuspid and first and third molar regions. Right: Recurrence of infection (March 8, 1938) around lower first molar and evidence of bone change between first and second molar

both preoperative and postoperative clinical observation they do not give the impression of being lesions of infectious processes. Such lesions definitely impress one as being necrotic tumor masses. I feel that it is more practical to classify them, along with osteitis fibrosa cystica and Paget's disease, as diseases caused by metabolic disturbances. If there is general objection to naming disorders after persons and preference toward giving descriptive titles, Schuller-Christian's disease could be called osseous eosinophilic lipogranuloma.

Regions of Occurrence

Schuller-Christian's disease may attack any of the bones of the body but occurs mainly in the flat bones, particularly the skull, pelvis and ribs. The long bones and jaws also may be involved. Although unusual the jaw bones may be the first to show signs of the disease. The dentist or oral surgeon may be confronted with it and should be on the alert for the condition. When suspicious of the disease one should call forth every available diagnostic aid because such lesions closely resemble osteogenic sarcoma, osteitis fibrosa cystica, acute granuloma, myeloma, cystic lesions and metastatic tumors.

Clinical Findings

To one who has been fortunate enough to study more than one case of Schuller-Christian's disease, the clinical findings become quite distinctive. The first symptoms are soreness, not pain, of the soft tissue and bone over the lesion without any clinical evidence of inflammation. Roentgenographic examination will show a rarefied re-

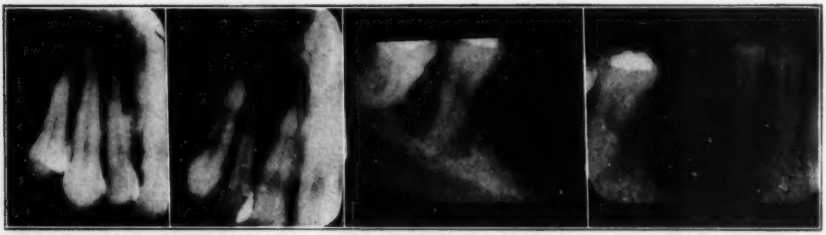

Fig. 2, Case 1.—Progressive destruction of bone (May 27, 1938) in both lower and upper left side. All teeth were vital and gum tissue was normal

Fig. 3, Case 1.—Roentgenogram of jaw (May 27, 1938) showing destruction of bone

gion within the bone. This may appear as a pyorrhea pocket or as a cystic formation. When it has the appearance of a deep pyorrhea pocket, there will be a definite opening along the side of the tooth root but no inflammation of the gum tissue. If this apparent pocket is between two teeth the interproximal gingiva will disappear as if it had been removed by electric cautery, but no inflammation will be noted. As the lesion develops and absorption of alveolus and bone progresses, the gum tissue appears to shrink and drop down with the loss of bone, but the gum remains firm and normal in color. There is no hypertrophy or red, inflammatory, spongy appearance as in most cases of gingivitis and stomatitis.

When all supporting alveoli have been destroyed the teeth naturally become loose and sore and must be extracted. If proper treatment is not given, the progress of disease is quite rapid, as will be shown in one case where a very small lesion developed to such an extent that the whole side of the mandible was involved within ten months. The impressive feature of such extensive lesions is the normal appearance of the overlying gum tissue, not unlike an edentulous ridge of several years' duration. In the case of the child who will be described, everyone thought it unusual that the 6 year molars were erupting at the age of 3 years. These teeth were not erupting, but the alveoli had been absorbed and the gums had dropped down to expose the occlusal surface without any inflammatory symptoms.

The bone defects within the mandible and maxilla appear to be confined to the cancellated bone structure, so there should be little fear of pathologic fracture or penetration of the antrum or nose. There is no bulging of the bones such as occurs with large cystic formations. In addition to the bone defects, patients with Schuller-Christian's disease are likely to have symptoms of exophthalmos and diabetes insipidus. All 3 of the patients to be reported on had the extreme thirst that goes with diabetes insipidus, and the little boy had the abnormal protrusion of the eyeballs common in exophthalmos.

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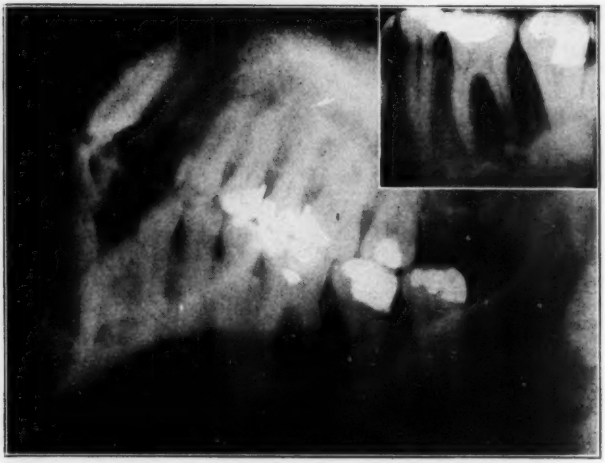

Fig. 4, Case 1.—Left: Pathologic condition of lower right molar region. Inset: Diseased regions between lower right first and second molars extending to apex of first molar

Roentgenographic Findings

Roentgenographic examination gives a variable picture. Usually it is similar to cases of myeloma, metastatic tumors and bone cysts but may at times resemble osteomyelitis. Like myeloma, metastatic tumors and osteitis fibrosa cystica, the osteolytic defects in Schuller-Christian's disease have a well defined outline, without the characteristic cystic margin of cortical bone as seen in odontogenic cysts. In the very early and superficial stage the picture is not unlike an extensive and solitary pyorrhea pocket or as a small area of acute inflammation when starting deep within the bone. As the lesion progresses it takes on an appearance similar to a cystic formation or a giant cell epulis. The bone regions look very much like an infected cyst or metastatic and epidermoid carcinoma of the jaw.

Pathologic Findings

The pathologic findings in Schuller-Christian's disease are not understood well. Biopsies require careful study of numerous sections, because too often fibrous and inflammatory tissues may hide the true picture, and a diagnosis of chronic inflammatory tissue is made. Sections do show much granulation and necrotic tissue with densely infiltrated plasma cells. Some may contain fibroblastic tissue and osteoblastic and phagocytic giant cells. But in addition to these, sections should show histiocytes, regions of eosinophilic infiltration and fat laden monocytes, for these are the real diagnostic findings for Schuller-Christian's disease.

Problems in Surgery

From a surgical standpoint the lesions of Schuller-Christian's disease are unique in character and consistency. The operator who has started with the belief that he was working with a cystic type of tumor will realize his error immediately when

opening the overlying gum tissue. Instead of a membranous sac filled with fluid, there will be a fairly dense mass filling the bone cavity which is grayish brown in color with occasional pinhead sized globules of yellow fat. It is impossible to curet or dissect the lesion en masse as it is necrotic and can be removed only in small sections. Also it seems impossible to remove the whole lesion because there is no demarcation between the soft tissue lesion and bone. One may curet far beyond the point where bone was indicated on the roentgenogram and still encounter soft tissue. Yet on pathologic examination these curettments will be filled with necrotic bone. The small masses curetted from these lesions resemble the granulomatous material removed from a tooth socket in which there is a small sequestrum of bone, but they are not hemorrhagic. Postoperative progress will appear normal, but later roentgenograms will show the contrary. It is the author's conclusion that surgery is of no avail in treating the lesions of Schuller-Christian's disease.

Roentgenotherapy is the only beneficial treatment. All workers seem to agree that such radiation will check the progress of these bone lesions for an extensive period and at times even stimulate regeneration.

The following three case reports should give a clear impression of the puzzling picture which Schuller-Christian's disease presents. The dentist or oral surgeon should be on the alert and use every diagnostic aid available when the disease is suspected.

Case 1

History.—A young married woman, aged 26 years, who had unusually good teeth with normal gum tissue, went to her dentist in August, 1936, complaining that the upper left second molar was tender to percussion. There was slight discomfort in this region but no real pain. The dentist found this tooth quite loose with a deep gingival pocket which indicated extensive alveolar destruction. The tooth was extracted and the postoperative condition was normal at first, but the socket never filled entirely.

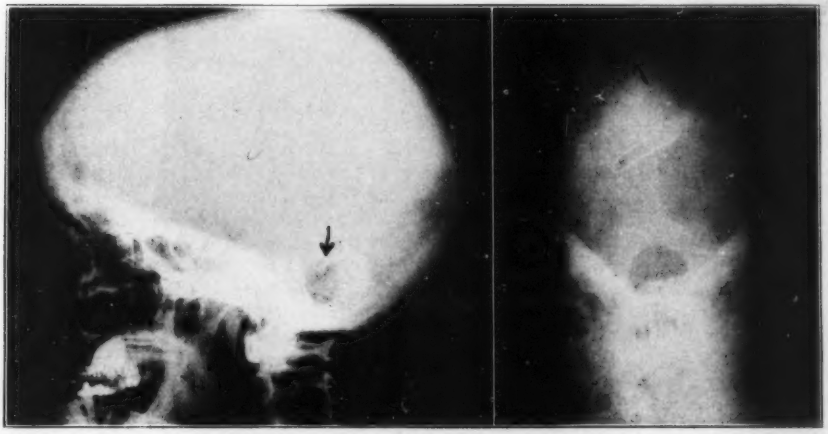

Fig. 5, Case 1.—Roentgenograms of the skull (March, 1941). Left: Necrosis of mastoid. Right: Area of bone destruction in left supra-occipital region

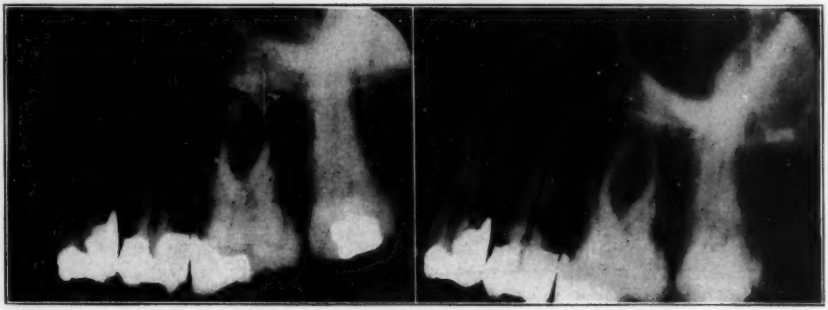

Fig. 6, Case 1.—Progress of disease in upper right bicuspid and molar region

Examination.—The patient was referred to the author in April, 1937, at which time a small curet could be passed deep into the upper left second molar socket and underneath the apices of the first molar. This, together with a roentgenogram of the area (Fig. 1, left and center), encouraged a diagnosis of a radicular cyst involving the second bicuspid and first and third molar regions.

Operation.—With the patient under block anesthesia, these teeth were removed and the whole region was curetted. I recognized at once an error in preoperative diagnosis, for the tissue removed from this bone cavity did not resemble a true cyst and the surrounding bone was not hard and smooth as it should be found. Pathologic study of the tissue removed showed nothing but chronic inflammatory tissue.

This large cavity was dressed with iodoform gauze, and the postoperative treatment consisted of changing the gauze dressing every forty-eight hours for about three weeks. Healing and filling in of the cavity appeared to be normal in every respect and the patient was discharged in a month.

Recurrence.—About a year later, March, 1938, the patient called again, complaining of slight discomfort around the lower left first molar. Clinical findings were negative because the teeth were vital and the gum tissue normal. However, a roentgenogram (Fig. 1, right) showed an area of apparent infection about the apex of the first molar as well as evidence of bone change between the first and second molars. A full mouth roentgenographic examination was negative for all other teeth and regions. The first molar was removed and the area curetted and cauterized. Healing appeared satisfactory except for slight discomfort around the adjacent lower teeth and the upper left first bicuspid.

Roentgenograms taken in May, 1938 (Figs. 2 and 3), showed progressive destruction of bone in both regions. It can be noted that there was definite bone change around the lower left second bicuspid and second molar and that the bone was destroyed entirely about the upper left central, lateral, cuspid and first bicuspid. It was at this time that I was at a loss to understand the cause of the unusual condition I was treating. It has been stated that all teeth were vital and the gum tissue normal in every respect, so no local cause for the abnormal bone disease could be found. The fact that it attacked both jaws within a short space of time seemed to rule out any true neoplasm or osteomyelitis. It seemed that the cause must be either some blood dyscrasia or some abnormal glandular function. The upper left first bicuspid was removed, the blood saved for bacteriologic examination and tissue removed for biopsy. Then the patient was referred to an internist for examination and blood test. After a physical examination and complete blood studies all findings were negative. There seemed to be no evidence of blood dyscrasia.

Bacteriologic Examination.—Examination of blood and smears revealed no pus cells, no yeast forms or fungi. Cultures yielded Streptococcus hemolyticus and Staphylococcus. Many sections

Fig. 7, Case 1.—Model for partial denture for maxilla

of tissue were examined by three different pathologists. These sections showed granulation tissue and regions of round celled and eosinophilic infiltrations; some showed endothelial and phagocytic giant cells. Some sections showed plasma cell, normal lymphocytes and fibroblastic tissue. However, all three pathologists arrived at one diagnosis—chronic and subacute inflammatory processes.

Treatment.—It can be seen that very little help was derived from physical examination and microscopic study. The work was continued blindly. In September, 1938, the patient returned complaining of some soreness in the lower right molar region. Roentgenograms (Fig. 4) showed definite pathologic regions between the first and second molars and extending down to the apex of the first molar. This first molar was removed but no curetting or further surgery was attempted.

After consultation with a roentgenologist it was decided to attempt roentgenotherapy to arrest the progress of this unusual bone disease. This proved to be very effective and caused prompt healing. In December, 1938, the upper first molar region became involved and was treated at once with roentgenotherapy.

After a series of roentgen treatments of the jaws it was believed that progress of the disease had been checked. I did not see the patient for more than two years. In March, 1941, I learned that she was in the hospital recovering from a mastoid operation. At biopsy the lesion of the mastoid was found to be almost identical with some of the specimens taken from the jaw. Roentgenograms of the skull at this time (Fig. 5) showed not only the necrosis of the mastoid but also an area of bone destruction in the left supra-occipital region. Upon these findings the pathologists gave a positive diagnosis of Schuller-Christian's disease.

When the patient called in January, 1945, roentgenograms (Fig. 6) showed marked progress of disease in the upper right bicuspid and molar regions. The teeth were removed and roentgenotherapy was given postoperatively. After healing of this region a partial denture was inserted. The model (Fig. 7) of the upper jaw for this denture shows very clearly how completely this disease destroys the alveolar portion of the jaws.

Results.—In January, 1947, the patient returned for examination. She admitted that she had neglected her mouth because of the expense of treating the skeletal bones. Clinical examination and roentgenograms (Fig. 9) showed typical lesions about the three remaining upper teeth, the lower left central, lateral and cuspid and the lower right cuspid. It seemed advisable to remove all remaining teeth. After this, radiation was used and full upper and lower dentures

were inserted. In spite of the loss of all teeth and involvements of many other bones, the patient appears to be in good physical condition after twelve years with Schuller-Christian's disease, and she is teaching school every day.

In the last twelve years following the onset of the disease in the jaw bones and the skull, the patient has had involvements of the pelvis, ilium, fibula, scapula and ribs. Figure 8 is a good example of this extension. But she has learned to recognize the characteristic soreness of the bone that goes with the lesion, and as new symptoms develop in various bones she is given roentgenotherapy. After treatment soreness is relieved and the progress is checked.

Case 2

History.—In April, 1945, a boy, aged 3½ years, was sent to the hospital for observation and diagnosis. His mother stated that her son had no physical complaints and had seemed normally healthy until a year previously when a slight swelling behind the right ear had been noticed, and the eyes appeared to bulge, with vision impaired. In August, 1944, he was taken to the family dentist because of a sore lower deciduous second molar. The tooth was free of caries but was removed, and after extraction the dentist stated that it had a granuloma attached. The dentist noticed that the lower six year molars were erupting, an occurrence unusual in a 3 year old child, but there was no definite pathology.

Examination.—Roentgenograms (Fig. 10) taken of the skull showed regions of bone destruction in the mastoid region, in the frontal and parietal bones, around each orbit, and definite

Fig. 8, Case 1.—Typical regions of Schuller-Christian's disease showing areas of destruction along crest of left ilium, above the hip sockets and in the right femoral shaft about 5 inches below the hip joint

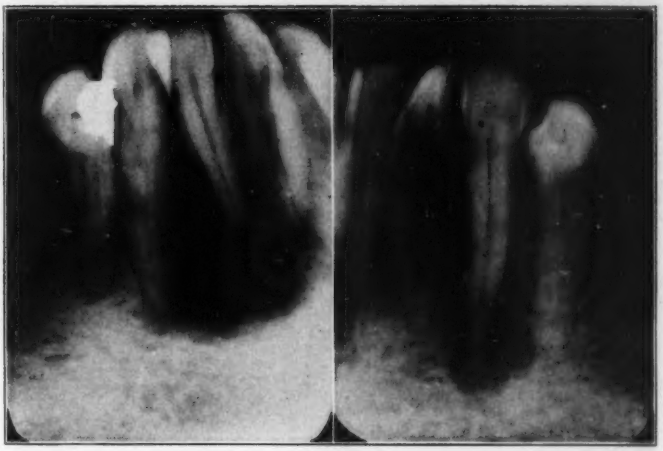

Fig. 9, Case 1.—Lesions around remaining lower teeth (January 10, 1947)

resorption of the alveoli of all deciduous molars. Examination of the mouth showed characteristics of Schuller-Christian's disease. The occlusal surfaces of the lower 6 year molars were visible, but they were not erupting. The absorption of alveoli had allowed the mucous membrane to drop down to these teeth. There was exposure of the roots of the upper left deciduous second molar. This tooth was removed and the area was curetted for biopsy.

Pathologic examination showed granulomatous lesions of soft tissue and bone similar to that seen in Schuller-Christian's disease, with collections of fat laden large monocytes and regions

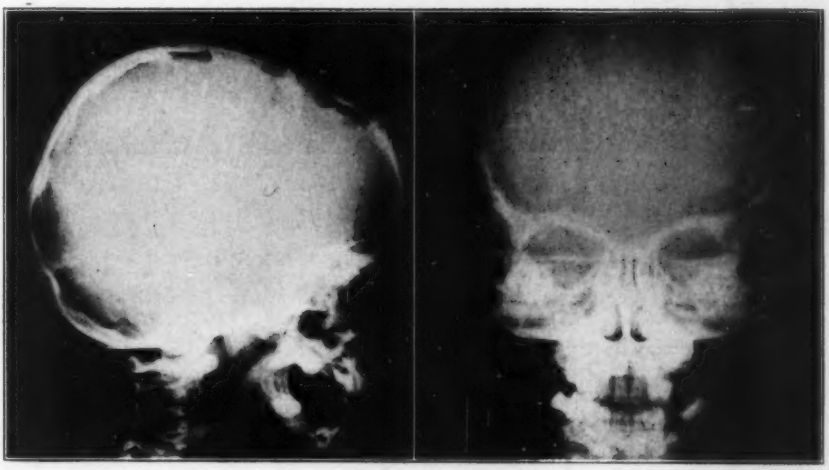

Fig. 10, Case 2.—Roentgenograms of skull showing areas of bone destruction in mastoid region, frontal and parietal bones, around each orbit and in molar regions on both sides of the mandible. Left: Lateral view. Right: Postero-anterior view

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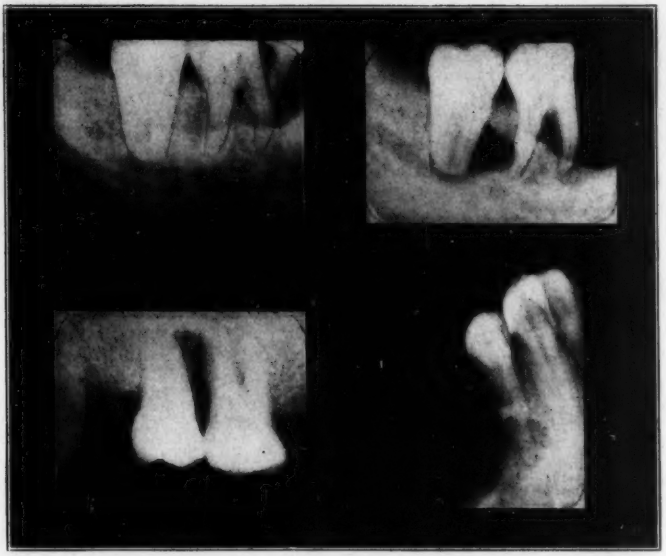

Fig. 11, Case 3.—Upper left: Alveolar resorption around a deciduous lower second molar (March, 1947). Upper right: Progressive cystlike region around first and second molars (August, 1947). Lower left: Deep pocket between upper left first and second molars (September, 1947). Lower right: Lingual pocket around lower left bicuspid (January, 1948)

of intense eosinophilic infiltration. From this report, the roentgenograms and clinical examination, a positive diagnosis of Schuller-Christian's disease was made and roentgenotherapy was ordered.

Treatment.—In the two years following these studies the child was given roentgenotherapy on the skull, 50 roentgens per treatment for a total of 150 roentgens. Roentgenograms of the skull taken in August, 1947, showed the many defects in the frontal bones, around the orbits and the jaw bones as previously but were less prominent than those seen in previous studies. From clinical observation I am sure there has been little progress of absorption of the jaw bones, and I feel confident that roentgenotherapy has held the disease under control.

Case 3

History.—A woman, aged 53 years, had a deciduous lower second molar which became quite tender. She consulted her dentist in March, 1947. A roentgenogram (Fig. 11, upper left) showed definite alveolar resorption around this deciduous molar which extended to the mesial of the permanent first molar. The deciduous molar was removed to relieve the condition, but in August the first and second molars and surrounding tissue became tender with no clinical evidence of pathology.

A roentgenogram of these teeth (Fig. 11, upper right) showed a progressive cystlike region around the first molar and one around the root of the second molar. These teeth were removed. In September the patient was referred to a periodontist because of trouble in the upper left molar region. A full mouth roentgenogram showed a deep pocket between the upper left first and second molars (Fig. 11, lower left) and a lingual pocket around the lower bicuspid (Fig.

Fig. 12, Case 3.—Regions of destruction which had developed by February, 1948. Left: Between lower right lateral incisor and cuspid. Center: In bone around upper first molar. Right: Around lower left cuspid

11, lower right), but the rest of the mouth was negative. It was decided to remove the upper left second molar before starting any treatment of the gums.

Diagnosis.—Because his treatments were not producing the usual results, the periodontist referred the patient to me for possible surgical treatment of pyorrhea. From the roentgenograms and clinical examination, I was suspicious of Schuller-Christian's disease, so I extracted the lower left first bicuspid and by curettment obtained several granulomatous masses for biopsy. Sections from these specimens were examined and compared with slides from the first and second cases presented, and a positive diagnosis of eosinophilic granuloma of bone was made.

Further roentgenograms of the jaws were taken which showed the start of a new area between the lower right lateral incisor and cuspid (Fig. 12, left) and progressive destruction of bone about the upper first molar (Fig. 12, center) and the lower left cuspid (Fig. 12, right). A full lateral jaw roentgenogram (Fig. 13, above) shows how extensive the destruction of bone throughout the lower left jaw had been.

The patient was referred to a roentgenologist for therapy. Studies were made of the ribs, the entire spine, pelvis, hips, femurs, lower legs and skull. All were negative except those of the skull which showed a small round area in the left side of the occipital bone about ¼ inch in diameter (Fig. 13, below).

Treatment.—Roentgenotherapy to the left side of the jaws and head was instituted, six treatments for a total of 1,160 roentgens. Follow-up roentgenograms taken three months after the start of treatment show no additional bone destruction; in fact, there was slight indication of regrowth of bone tissue in the lower jaw.

Theories of Various Workers

Farber, Jaffe and Lichtenstein seem to agree that eosinophilic granuloma of bone, Letterer-Siwe's disease and Schuller-Christian's disease constitute different expressions or phases of the same basic disorders. They find that in the early stages of Letterer-Siwe's disease one may find destructive skeletal lesions resembling those of eosinophilic granuloma of bone, and the lesions of bone in Schuller-Christian's disease originally may resemble those of eosinophilic granuloma of bone. Schuller-Christian's disease represents the basic disorder in its most chronic and, on the whole, most miscellaneous form and should be expected to show, as an anatomic prerequisite, at least some lesions which have been scarified, collagenized and lipidized and so are classified as lipogranuloma.

From a practical and clinical standpoint, it is difficult to understand how it is possible to determine a dividing point for these three stages of disease. In Figures 1, right, and 2, extreme right, the rapidity of growth from a primary lesion to extensive bone destruction in ten weeks' time can be observed. In Figure 11, upper left

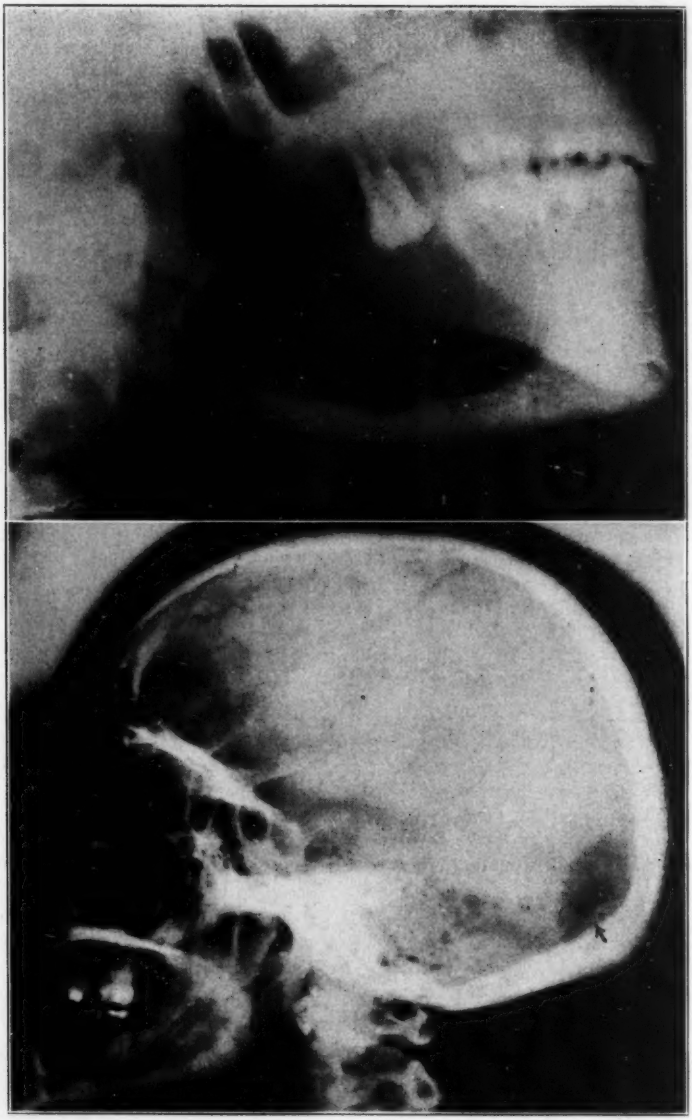

Fig. 13, Case 3.—Above: Extensive destruction of bone throughout lower left jaw (February, 1948). Below: Small area of destruction on left side of occipital bone (March, 1948)

and right, no lesion was shown around the last molar in March, but an extensive one was present in August. It shows very clearly that such lesions originate and progress too rapidly to divide into three separate phases. The facts are that such lesions are not likely to be discovered until the patient has symptoms of soreness of the part, at which time the disease is advanced into the stage of Schuller-Christian's disease. Therefore, it is impractical and confusing to divide such a disturbance into three phases; I wish to go on record as favoring the continued use of the name Schuller-Christian's disease for all such phases. If, as stated before, there is objection to naming disorders after persons, all phases can be covered with the name osseous eosinophilic lipogranuloma.

In 1932 Schaefer[7] presented a case which resembled closely the first case reported in this paper. His roentgenograms show many different lesions in apparently all three phases within the same jaw bone. It is interesting to note that he stresses that the patient's teeth were in excellent condition and that the gums appeared normal and healthy. He also points out that the most effective treatment is roentgenotherapy to the affected regions. Many other forms of treatment have been tried without universal success. All experienced observers agree that roentgenotherapy is the only positive treatment.

Summary

Schuller-Christian's disease is a rare disturbance of the skeletal bones, principally the flat bones and bones of the skull. Although it is unusual, the jaw bones may be the first to show destructive lesions, so dentists should be on the alert.

The regions of bone destruction very closely resemble those seen in osteitis fibrosa cystica, myeloma, cystic regions and metastatic tumors, but swelling and inflammatory reactions are absent. The most striking symptoms are slight soreness of the tissues over the lesion without any evidence of inflammation of the surrounding tissue.

Pathologic study for biopsy requires thoroughness. The first impression gives a picture of chronic inflammatory tissue, but more careful study will show histiocytes, regions of eosinophilic infiltration and fat laden monocytes. These are the real diagnostic findings.

Various forms of treatment have been tried without success, and surgical interference is of no avail. Roentgenotherapy is the most positive form of treatment and will check the progress, at least for a time, of the destructive lesions of Schuller-Christian's disease.—*921 State Tower Building.*

7. Schaefer, J. E., and Williams, P. E., Xanthomatosis (Schuller-Christian's Disease). *D. Cosmos* 74:879 (September) 1932.

Acute Secondary Parotitis. W. A. Altemeirer, in *Surgery* 20:191 (August) 1946.

Acute secondary parotitis may be treated: (1) symptomatically, by the use of massive hot moist compresses to the involved area; (2) by surgical drainage of localized abscesses; (3) by roentgen-ray or radium, and (4) by repeated dilatation of Stensen's duct. The author has found the systemic administration of large doses of Lugol's solution and in an occasional case the use of penicillin very effective. Lugol's solution in massive doses has the following action: (1) bacteriostasis and (2) stimulation of salivary secretion.—*Quart. Rev. Otorhinolaryng. & Br.-Esoph.* 6: March, 1947.

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FRACTURES OF THE MANDIBULAR SYMPHYSIS WITH REPORTS OF CASES

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THE MANAGEMENT of fractures through the symphysis region of the mandible is occasionally quite a complicated procedure. The proper manipulation of fragments and the adequate fixation of component parts of the fracture frequently tax the ingenuity and dexterity of both the oral and the plastic surgeon. The combination of two technics, internal wire fixation with a Kirschner type pin and an arch bar of the Risdon variety, has been used quite effectively in the treatment of the more complicated fractures of the symphysis.[1-3]

Internal Wire-Arch Bar

The internal wire-arch bar provides an easy and satisfactory method of fixation where there is marked displacement, overriding of the fragments or comminution of the bone. It is especially advantageous in patients whose general condition makes it unsafe to wire the mouth in occlusion (Fig. 1, left, and 2, left). The method provides adequate stabilization without interdental fixation of the maxillary and mandibular dental arches.

Cerebral trauma and other concomitant injuries accompanied by nausea and vomiting or obstruction of the airway necessitate the mouth being open. The danger of aspiration of vomitus following a general anesthetic required by a young or uncooperative patient may be avoided completely by the open technic (Figs. 1 and 2). Intra-oral injuries also may demand care while the fracture is healing. In the child whose teeth make interdental wiring impractical (Fig. 1, left), in the old edentulous patient (Figs. 3 and 4) and in those instances where interdental wiring has failed, the internal wire fixation and arch bar stabilization are wholly gratifying.

Procedure

The procedure is carried out best with bilateral mandibular blocks and infiltration of the soft tissues through which the pin is to be introduced. However, in smaller children, uncooperative adults and in those whose general condition does not permit work under regional anesthesia, a general anesthetic may be administered with impunity.

At operation the oral mucosa and teeth are cleansed thoroughly. The skin is prepared with iodine and alcohol; the patient is draped. The fracture is manipulated and the fragments placed in their proper alignment. Then the best position and most satisfactory direction for inserting the pin is determined.

A small stab incision is made in the skin and subcutaneous tissues. The periosteum is denuded from the bone at the point where the pin is to enter. A stainless steel

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The authors wish to acknowledge the cooperation of James Barrett Brown, M.D., and L. W. Peterson, D.D.S., in preparation of this paper.
1. Brown, J. B., and McDowell, Frank, Internal Wire Fixation for Fractures of Jaw. *Surg., Gynec. & Obst.* 74:227 (February 1) 1942.
2. Brown, J. B., and McDowell, Frank, Internal Wire Fixation of Jaw Fractures. *Surg., Gynec. & Obst.* 75:361 (September) 1942.
3. Risdon, Fulton, The Treatment of Fractures of the Jaws. *J. Canad. D. A.* 6:471 (October) 1940.

Kirschner wire of sufficient rigidity to provide the necessary support (gage .035 to .045) is inserted by means of an ordinary motor-driven bone drill. After the pin has engaged and passed through both fragments and lies just beneath the opposite skin surface, another small incision is made to emit the pin. The pin is cut on each side and should extend only about 2 cm. beyond the surface. Small corks are placed on the exposed portion of the Kirschner wire for protection of both the pin and the soft tissue[2] (Figs. 1, left, and 6, below).

After the pin has been positioned satisfactorily, the Risdon type bar is applied to the lower arch (Figs. 4 and 6, below). A piece of No. .025 stainless steel wire, 14 inches long, is placed around the second molars and with equal lengths of the wires projecting from the corners of the mouth. Each wire is twisted on itself until it is tight on the teeth. Then each twisted wire is carried forward to the midline. Here they are twisted tightly together (Fig. 2, right). Of course, the placement of the bar may have to be modified on occasion because of missing teeth. The arch bar is anchored to other teeth by individual wires. At the termination of the procedure, a bulky pressure dressing is applied neatly and accurately.[3]

It must be emphasized especially that interdental wiring is a very essential part of the treatment in those cases with multiple fractures of the mandible and other facial bones. However, the internal wire-arch bar procedure may be combined easily with interdental wiring (Figs. 5 and 6, above). It also may be used so effectively as to permit the delay of the application of interdental wires until the patient's general condition permits closure of the mouth with safety. In the edentulous patient, the Risdon arch bar cannot be used, but the pin provides very adequate fixation in many cases and certainly offers the simplest solution for the treatment of such fractures (Fig. 3).

One of the most important features of the combined arch bar and Kirschner wire is the absolute stabilization of the fragments in all directions. The Kirschner wire provides fixation in the vertical and anteroposterior planes. The arch bar prevents separation in the lateral or horizontal planes.

The materials used in this procedure are universally available. The instruments

Fig. 1, Case 1.—Left: Postero-anterior roentgenogram showing reduction of fragments and Kirschner pin in position. Right: Appearance of patient with mouth in closed position. Protective corks and bandage removed from pin

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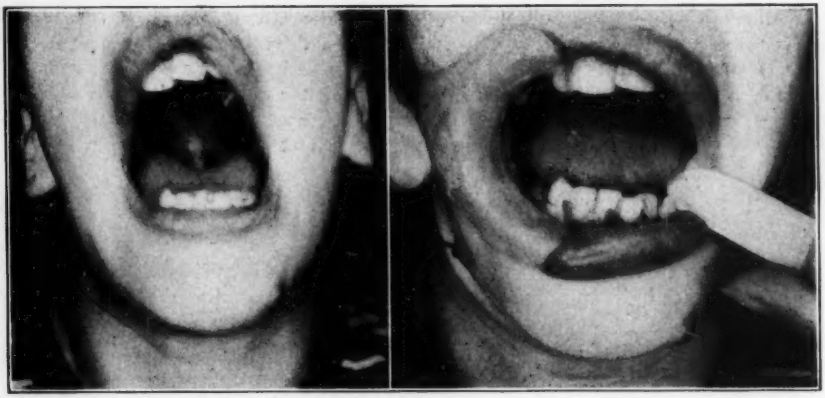

Fig. 2, Case 1.—Left: Appearance of patient with mouth open. Comparison with Figure 1, right, shows extent of movement of mandible. Right: Intra-oral photograph demonstrates the modified Risdon arch wiring which extended from cuspid to cuspid

required are standard operating room equipment. In this way complicated dental appliances which are constructed for the individual case are eliminated.

The patient finds this type of fixation comfortable. Oral hygiene is maintained easily. This method of immobilization allows movement of the jaw; consequently, the dangers from nausea, vomiting and inadequate airway are eradicated. This is important especially in patients who are transported by sea and air. It is highly useful when it is necessary to administer a general anesthetic for further surgery (Fig. 2, left).

Proper reduction before placing the pin is absolutely necessary; little or no change can be made following its insertion. It is important to avoid the nerves and roots of the teeth. Furthermore, the pin must be placed sufficiently high in the bone to avoid cross fracture of the mandibular cortex. Fortunately, this is accomplished quite easily in the symphysis region because of the relatively high position of both teeth and nerves. Heat necrosis to bone does not occur if the motor drill is run at a moderate speed.

Postoperative Treatment

Following the operative procedure, the patient is placed on antibiotics; penicillin 50,000 units every three hours or procaine penicillin (Duracillin[4]) in sesame oil 300,000 units daily. A liquid diet is advisable for the first seven to fourteen days, after which time the patient can change to a soft diet. Although neither the wires nor the pins require constant adjustment as in many types of fixation, it is advisable to see the patient once a week. The pins usually can be removed after the third or fourth week; the arch bar remains for an additional one or two weeks.

Occasionally small abscesses occur in the region of the fracture site as in any other method of treatment, but no extensive osteomyelitic processes have been

4. Duracillin is the trade mark used by Eli Lilly & Co. to designate its brand of procaine penicillin.

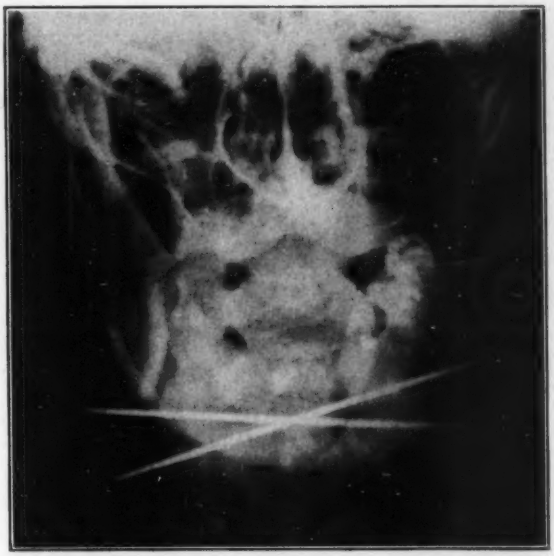

Fig. 3, Case 2.—Postero-anterior roentgenogram showing reduction of fragments and the position of Kirschner pins in a mandible which, for all practical purposes, is edentulous

observed. The pins always are found to be tight at the time of removal which suggests little or no reaction of the surrounding bone. There have been no cases of nonunion observed.

Reports of Cases

Case 1

History.—A 12-year-old white boy was kicked in the chin by a horse. The boy apparently was not unconscious at any time following the injury.

Physical Examination.—The patient was admitted to the hospital on May 2, 1948. There was a deep laceration of the soft tissue of the chin which was approximated poorly by a metal skin clip. Only a moderate amount of swelling and very little trismus were present. Crepitation was noted in the symphysis.

Roentgenographic Examination.—Roentgenograms of the mandible revealed a fracture of the symphysis approximately 1.5 cm. to the right of the midline.

Operation.—On May 5, 1948, endotracheal anesthesia was administered. The patient was prepared and draped. Adjustment of the fragments was completed. Then the Kirschner wire was placed through the fragments from right to left. A modified type of Risdon arch bar was applied from cuspid to cuspid. This was done because of a premature loss of deciduous molars and the permanent first molars. The fragments were stabilized well in all directions.

Postoperative Roentgenograms.—Examination showed fragments in good position as seen in Figure 1, left.

Postoperative Course.—The patient was dismissed from the hospital on May 10 and was seen once weekly. The postoperative course and progress were uneventful. The bar and pin were removed at the end of the fifth postoperative week. (Figs. 1 and 2).

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Fig. 4, Case 3.—Postero-anterior roentgenogram showing immobilization of fragments with the Kirschner wire and the Risdon external arch wiring

Case 2

History.—A 25-year-old white woman was in an automobile accident on July 9, 1946. Her face apparently struck the dash panel of the car. She had experienced pain and swelling in the region of her chin since the injury. There was no period of unconsciousness following the accident.

Physical Examination.—The patient entered the hospital on July 11, two days after the accident. There was a laceration of the chin. Several of the lower anterior teeth either were chipped or broken at the gum line. Crepitation in the region of the symphysis was apparent. There was also a complete separation of the anterior alveolar bone. The upper arch was edentulous, and there were only scattered lower teeth remaining. Other physical findings were negative except for minor contusions of both knees.

Roentgenographic Examination.—There was a fracture through the mandible in the region of the symphysis; the fragments were in good position. There was also a fracture of the left condyle with slight medial displacement.

Operation.—The patient was prepared, draped and general anesthesia was administered. The five lower anterior teeth were removed, the comminuted alveolar bone was dissected free from the other fragments of the mandible and the tissue was closed. The fragments at the symphysis were reduced and immobilized by passing two Kirschner wires transversely across the symphysis. A pressure and supporting bandage then was applied (Fig. 3).

Postoperative Roentgenograms.—Roentgenograms of the mandible showed the fracture at the symphysis to be in good position (Fig. 3). The fragments were held in position by pin fixation. No significant change was seen in the left condylar fracture.

Postoperative Course.—The patient was dismissed from the hospital on the sixth postoperative day and then was seen once a week. The course was uneventful, and the pins were removed at the end of the sixth postoperative week.

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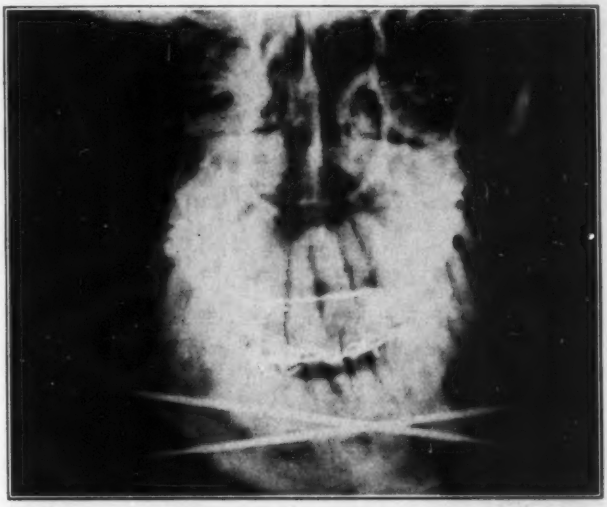

Fig. 5, Case 4.—Postero-anterior roentgenogram showing Kirschner pins in position and maxillary and mandibular Risdon arch bars

Comment.—Since the Kirschner type pin was to be used and the anterior teeth would have to be removed at a later time, it was advisable to remove these teeth along with the comminuted alveolar bone. Thus, the chance of sequestrum formation and osteolytic changes which might extend to the pins were avoided.

Case 3

History.—A 41-year-old white man sustained injury during an attack by an unknown assailant on June 30, 1946. The patient was unconscious for a short time following the attack.

Physical Examination.—The patient was admitted to the hospital on July 2, 1946. Swelling and tenderness of the soft tissue of the chin and some trismus were noted. Crepitation was felt between the right mandibular lateral and cuspid teeth. There was some displacement and separation of the fragments. Other physical findings were negative.

Roentgenographic Examination.—There was a fracture of the mandible in the symphysis region to the right of the midline. Separation of the fragments was quite evident. There was also a fracture of the neck of the left condyle, but no displacement was apparent.

Operation.—On July 3 the operation was performed using a local anesthesia of 2 per cent procaine. The patient was prepared, draped and anesthetization of the tissues was carried out. The fragments were reduced and a Kirschner wire was placed through the symphysis engaging both fragments. A Risdon arch bar was placed on the lower arch. A drain was placed at the fracture site.

Postoperative Roentgenograms.—Roentgenograms of the mandible showed the fragments to be reduced and held in good position by a Kirschner wire and a dental arch bar (Fig. 4).

Postoperative Course.—The drain was removed on the fourth postoperative day, and the patient was discharged immediately. The patient was seen once weekly, and the bar and pin were removed on the sixth postoperative week.

Case 4

History.—A white man, aged 23 years, sustained injuries in an automobile accident in which

he was driving. His face apparently had struck the steering wheel or the windshield. He had been unconscious for a few moments following the accident.

Physical Examination.—The patient was admitted to the hospital on January 1, 1948, twelve hours after the accident. The patient had multiple superficial lacerations of the lips and a deep laceration of the soft tissues of the chin. There was a depression of the malar bone and infra-orbital ridge and proptosis of the right eye. Both halves of the palate moved independently of each other. There was a fracture of the symphysis of the mandible with marked displacement of the fragments. Bleeding from both nostrils and the right ear was evident. The patient showed signs of mild concussion.

Roentgenographic Examination.—Roentgenograms of the facial bones and mandible showed comminuted fracture at the symphysis and a fracture of the left condyle. There was a horizontal fracture of the maxilla and right malar bone with involvement of the right antrum and the floor of the right orbit.

No attempt at reduction of the fractures was made until the sixth day of hospitalization because of the continued otorrhea from the right ear. The jaws were supported during this interval by the use of a head bandage which, of course, was not satisfactory but did add to the comfort of the patient.

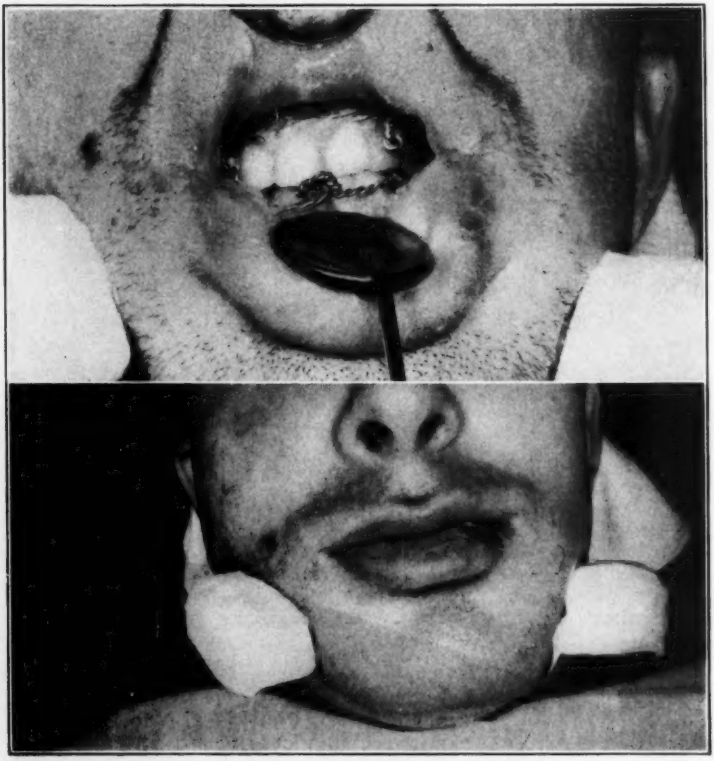

Fig. 6, Case 4.—Above: Oral photograph demonstrating the maxillary and mandibular Risdon arch bars in position. Below: Photograph showing the protection of Kirschner pin with corks over which a small bandage is placed

Operation.—The patient was operated upon on January 6, under an anesthetic of thiopental sodium by induction and ether endotracheally. He was prepared and draped, and a Risdon bar was placed on both upper and lower arches but not completely tightened. Then the right antrum was entered, and after manipulation and reduction of the right malar bone and wall, the antrum was packed with 2 inch iodoform gauze. The mandibular fracture was reduced further, and two Kirschner pins were placed through the symphysis region. The Risdon bar was tightened further and interdental wires closed the jaws in good position.

Postoperative Roentgenograms.—Roentgenograms of the facial bones showed reduction of the maxilla and malar bone with packing in the right antrum. The symphysis fracture was reduced and in a satisfactory position. Two Kirschner wires can be seen passing through fragments of the symphysis region, and dental arch bars should be noted on the upper and lower teeth.

Postoperative Course.—The postoperative course was uneventful, but the patient was not discharged until January 2, 1948. The antral pack was removed on the tenth postoperative day. The patient was seen once weekly, the pins were removed on the seventh postoperative week and the arch bars on the eighth week (Figs. 5 and 6).

Repair of Bone Loss and Fractures of the Face and Mandible with Cancellous Bone Chips. By E. W. Pickard, in *Canad. M. A. J.* 57:556 (December) 1947.

Pickard describes his experiences with 10 cases of non-union of the mandible treated by open operation, and bone grafting, with cancellous bone chips from the iliac crest.

These non-unions were in various positions in the mandible. Preliminary adjustable splinting was provided by means of cap splints applied to the upper and lower teeth, with locking devices. The operation is carried out through an incision through the skin at the lower border of the mandible. Bone is removed from the fracture site; the fractured fragments are placed in position to give satisfactory occlusion, and the cancellous bone chips, which are cut from the iliac crest by means of osteotomes, are put in position. The chips which were used measured about 1 cm. by ½ cm. by 2 mm. A plate of bone is also taken which is used to bridge the gap and overlap each fragment by about one centimeter.

The chips are packed irregularly, and the soft tissues sutured over them to hold them in position. A drain is left in position in some patients to guard against the formation of an hematoma. Despite the presence of infection in some of the cases, it was not necessary to remove this type of bone graft, and union eventually took place.—*Plast. & Reconstr. Surg.* 3: May, 1948.

POSTGRADUATE COURSES

The College of Dentistry, The Ohio State University, is offering seven postgraduate courses, five days each in length, during the autumn quarter of 1948. Those courses of interest to the oral surgeon are: anatomy of the head and neck (L. F. Edwards), September 20 to 24; x-ray (H. D. Spangenberg Jr.), October 11 to 15; oral pathology (H. B. G. Robinson), October 18 to 22; and oral surgery (D. P. Snyder), December 6 to 10.

The courses in anatomy, oral pathology, oral surgery and x-ray will be offered again during the winter and spring quarters.

The fee for the oral surgery course is $100; the fee for each of the other subjects is $50. Veterans are eligible to take this work under the G.I. Bill of Rights.

Further information and application forms may be obtained from the Postgraduate Division, College of Dentistry, The Ohio State University, Columbus 10, Ohio.

UMI

CENTRAL GIANT CELL TUMORS OF THE JAWS

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Los Angeles

THE central giant cell tumor which occurs in the jaws is the same lesion as that found in the long bones of the skeleton. The tumor is a different entity from the peripheral giant cell tumor of the gums, the so-called epulis. They are found to be identical only on examination of a microscopic section.

Literature

Warren[1] described giant cell tumors of the maxilla as well as benign central tumors of the femur and tibia. However, credit goes to Nélaton[2] for the first extensive investigation concerning giant cell tumors of bones, and he pointed out their benign character. One of his illustrations showed a lesion in the maxilla involving the canine fossa and maxillary sinus. Since that time the literature has become quite extensive. There have been, however, few articles that have restricted their content solely to central giant cell tumors of the jaws. This tumor has been known by many names, such as myeloid sarcoma, giant cell sarcoma, giant cell tumor, osteoclastoma, Braun tumor and others. From 1870 to 1910 the terms myeloid sarcoma and giant cell sarcoma were in common usage; consequently the treatment used was of a radical nature. In 1910 Bloodgood[3] pointed out the benign character of this tumor and changed the name to benign giant cell tumor. This now is accepted universally.

One group of investigators explain the pathogenesis of the giant cell tumor as a true neoplasm. Ewing,[4] Bloodgood,[5] and Geschickter and Copeland[6] are a few of the men who believe in this theory. On the other hand, Barrie,[7] Mallory,[8] Lubarsch[9] and Konjetzny[10] thought they were able to demonstrate that this lesion is an inflammatory and resorptive process. Barrie[7] considered it as a hemorrhagic osteomyelitis with a pathologic picture of a regenerative or granulative process whose end effect is the restoration of regions already destroyed by some injurious factor. Lubarsch[9] defined this lesion as a process of resorption and organization of focal hemorrhage and not a true neoplasm. Konjetzny[10] showed that intermedullary hemorrhage causes a reactive proliferative process which consists histologically of all elements encountered in giant cell tumors.

Geschickter and Copeland,[6] on the other hand, brought forth a theory which deserves special consideration. To them the tumor was the end result of an imbalance of two normal repair processes, one an unusual increase in osteoclastic proliferation and the other a diminution or a suspension of new bone formation in an

From the Department of Oral Histopathology, School of Dentistry, University of Southern California, Los Angeles.

1. Warren, J. C., 1837. Cited from Ewing, James, *Neoplastic Diseases*. Philadelphia: W. B. Saunders, Third edition, 1931.
2. Nélaton, Auguste, Tumeurs benignes des os ou tumeurs a myeloplaxes. Paris, 1860.
3. Bloodgood, J. C., Benign Bone Cysts, Osteitis Fibrosa, Giant Cell Sarcoma and Bone Aneurysms of Long Pipe Bones. *Ann. Surg.* 52:145, 1910.
4. Ewing, James, Review and Classification of Bone Sarcomas. *Arch Surg.* 4:485 (May) 1922.
5. Bloodgood, J. C., The Conservative Treatment of Giant Cell Sarcoma with the Study of Bone Transplantation. *Ann. Surg.* 56:210, 1912.
6. Geschickter, C. F., and Copeland, M. M., Tumors of the Giant Cell Group. *Ann. Surg.* 21:145 (July) 1930.
7. Barrie, G., Multiple Hemorrhagic Foci in Bone. *Ann. Surg.* 71:581 (May) 1920.
8. Mallory, F. B., Giant Cell Sarcoma. *J. M. Res.* 24:463, 1911.
9. Lubarsch, O., Lokalisierte Osteitis Fibrosa. *Arch. f. klin. Chir.* 121:147, 1922.
10. Konjetzny, G. E., Die Sogennante "Localized Osteitis Fibrosa." *Arch. f. klin. Chir.* 121:567, 1922.

injured cortex. The abnormal osteoclastic proliferation may occur as a phase in the histogenesis of intracartilaginous bone. In support of their theory, they contended that there occurs an embryologic proliferation of giant cells during the shift in ossification from cartilage to bone, and that giant cell tumors develop at the site where bone is formed from cartilage. Kolodny,[11] in an excellent review of the literature, stated that "although opinion is not yet settled as to whether the lesion is a true neoplasm or merely an inflammatory process, the trend of opinion in recent years has inclined toward the inflammatory."

Study of Cases Reported Since 1900

An attempt was made to collect and study all cases of central giant cell tumors of the jaws reported in the literature and those available from the Department of Pathology, School of Medicine, and the Department of Oral Pathology, School of Dentistry, University of Minnesota. Only those cases were selected from the literature in which case histories were available for analysis. It seemed that this material offered a representative picture of the frequency of these tumors because it included all cases reported since 1900.

Central giant cell tumors of the jaw are very uncommon. It was found that the incidence of central giant cell tumor was slightly less than 0.01 per cent of the total admissions (67,833) to the dental clinics at the School of Dentistry, University of Minnesota. This figure was based on the number of patients registered in the outpatient clinic in a ten year period (1931-1941). There were 2,134 oral lesions treated in the Department of Oral Pathology in this period, and of these only 9 were found to be central giant cell tumors (0.43 per cent). In the Department of [General] Pathology where a total of 28,911 operative specimens were examined, only 0.006 per cent were giant cell tumors of the jaws.

The occurrence of giant cell tumors of the jaws in relation to giant cell tumors of the rest of the skeleton was 11.3 per cent (Table 1). Simmons[12] found only 10 cases in the jaw out of his collection of 116 giant cell tumors. Geschickter and Copeland[13] were able to collect 20 cases (9 per cent) in their series of 226 giant cell tumors.

11. Kolodny, Anatole, *Bone Sarcoma.* Chicago: Chicago Surgical Publishing Company, 1927.

12. Simmons, C. C., Malignant Changes Occurring in Benign Giant Cell Tumors of Bone. *Surg., Gynec. & Obst.* 53:469 (October) 1931.

13. Geschickter, C. F., and Copeland, M. M., Osteitis Fibrosa and Giant Cell Tumor. *Arch. Surg.* 19:169 (August) 1929.

Table 1.—Incidence of giant cell tumors of the jaw in relation to giant cell tumors found elsewhere in the body

Investigators	Number of tumors	Number of jaw tumors	Percentage
Simmons[12]	116	10	8
Geschickter and Copeland[13]	226	22	9
Mayo Clinic[14]	84	16	19
Christensen[15]	362	41	11
University of Minnesota	28	4	14
Total	816	93	11.3

In the 84 cases collected at the Mayo Clinic, Kirklin and Moore[14] reported 16 cases located in the jaws. Christensen,[15] in his analysis of 362 cases, found 41 lesions (11 per cent) situated in the jaw. In the series at the University of Minnesota, 4 cases out of 28 giant cell tumors were found in the mandible or maxilla.

In the present study only 75 cases were collected; 60 from the literature and 15 from the material at the University of Minnesota. These tumors were found to occur in women more frequently than in men. Forty (62.1 per cent) of the cases were found in women; 29 (37.9 per cent) in men.

Occurrence According to Age

It is agreed generally that giant cell tumors are lesions of early adult life. Geschickter and Copeland[13] estimated that 40 per cent of all their cases appeared in the third decade of life. Simmons[12] concurred with them on the age distribution. Kolodny,[11] on the other hand, found them to occur most commonly between the ages of 15 and 25. The age incidence of the material studied is illustrated in Figure 1. These tumors appeared most frequently in the jaws during the second decade, at which time 40 per cent of the growths were found. They occurred a decade earlier than did the giant cell tumors of the long bones as estimated by Geschickter and Copeland.

In the cases found to exist in the first decade, 5 were in children 6 years old or less. The oldest patient in the series studied was 59 years old. There was no correlation found between age and sex, although in the second decade group, 19 cases were found in men and only 10 in women. In the other decades the women predominated.

Christensen,[15] in a general discussion of giant cell tumors of bone, reported 21 cases occurring in the mandible and 20 in the maxilla. In Kirklin and Moore's series at the Mayo Clinic, 9 occurred in the maxilla and 7 in the mandible. In the material studied for this report, 50 patients had tumors in the mandible and 25 in the maxilla. In the 25 cases involving the maxilla, 16 tumors were found in the anterior region mesial to the first bicuspid. They all involved the antrum. The remaining cases were located throughout the bicuspid and molar regions. There appeared to be no side predilection. The tumors of the mandible were found throughout the jaw with equal frequency (Table 2).

Geschickter and Copeland stated that the lower jaw was the most frequent site of these tumors in the skull. They also concluded that these tumors were located in the areas of cartilaginous nuclei for the mandible; namely, near the symphysis and in the region of the ramus where the osseous structures are derived from cartilage. They based their opinion on the conception that these tumors arise from the embryologic proliferation of giant cells during the shift from cartilage to bone.

This theory can not be accepted by us because, in the cases studied, typical central giant cell tumors developed in the bicuspid and molar regions with the same frequency as those which developed from the symphysis and ramus. The tumors located in the bicuspid and molar regions were limited to these regions only and, consequently, could not have had any direct relation to the cartilaginous nuclei at the symphysis and ramus.

Symptoms

A few symptoms are produced by giant cell tumors. The sequence given by Geschickter and Copeland is trauma, pain and finally a fracture of bone occurring

14. Kirklin, B. R., and Moore, C., Roentgenographic Manifestations of Giant Cell Tumor. *Am. J. Roentgenol.* 28:145 (August) 1932.

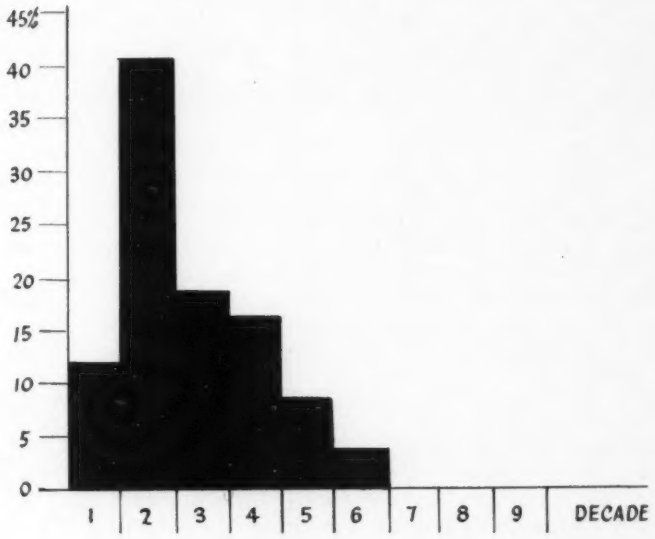
15. Christensen, F. C., Bone Tumors. *Ann. Surg.* 81:1074 (June) 1925.

within a period of two to fourteen months. Kolodny also believed that the giant cell tumor was of slow growth and of long duration. In the cases studied, there was a history of trauma accompanying the development of the tumor. Trauma was of the nature of a definite blow on the jaw or the sensation on extraction of a tooth. Pain was not a definite feature in the majority of the histories studied.

The duration of the symptoms varied from two weeks to five years. Figure 2 shows that 29.6 per cent of the patients had symptoms five to eight months before consulting the surgeon for treatment. There was 18.5 per cent (10 cases) with symptoms of more than twenty months' duration. As a result of slow growth and absence of pain, the bone destruction was so great that in some cases it led to a pathologic fracture.

Examination

The destructive character of this lesion is well illustrated in the roentgenographic



Decades	Males	Females	Total
1	4	5	9
2	19	10	29
3	3	10	13
4	3	9	12
5	0	6	6
6	0	3	3
Total	29	43	72

Fig. 1.—Age incidence of giant cell tumors studied

Table 2.—Location of giant cell tumors of the jaws

Side	Anterior 1-2-3	Bicuspid 4-5	Molar 6-7
Maxilla	16	5	3
Right (13)	9	3	1
Left (11)	7	2	2
Mandible	18	21	15
Right (19)	7	6	6
Left (35)	11	15	9

examination. In the maxilla there appear multiple irregular osteolytic foci with a margin denoting their infiltrating character. In the mandible a well demarcated cystic region generally forms, of irregular outline and trabeculated. The teeth may be displaced and often may show resorption.

The growth is not infiltrative in nature but expansive. The gingival tissue has a darker color than normal. Where the tumor has extended through the cortical bone, the underlying bone in some cases may present a cystlike softening on palpation. The teeth sometimes may be spaced and loosened from the destruction of the underlying bone and cementum. Finally, they may fall out. A typical lesion consists of solid portions as well as small cystic regions. The solid portions are friable, crumbly, granular masses varying in color from yellow to dark red. The tissue is very vascular, soft and readily oozing. The tumor may bleed when cut. It even may be encapsulated in some instances by fibrous tissue.

The growth is found to be composed of large, multinucleated giant cells loosely embedded in a stroma of small, rounded spindle or polygonal cells with large vesicular nuclei. The stroma varies considerably in appearance. It may be dense as scar tissue, or it may be cellular with numerous nuclei. Stimulation by infection, hemorrhage, fracture or roentgenotherapy commonly increases this cellular or sarcoma-like appearance. The giant cells are large, opaque, acidophilic cells containing from 15 to 60 nuclei. These are arranged loosely and centrally rather than ringlike and peripherally as in the Langhans type of giant cells.

Metastases from giant cell tumors have been reported. Simmons[12] reported 4 cases in which death occurred from metastases. Coley[16] reported 50 cases, 9 of which metastasized. It seems possible that many of these primary tumors were osteogenic sarcoma containing giant cells rather than typical central giant cell tumors. In the material studied, not one of the cases showed metastases.

Weinmann and Sicher[17] discuss giant cell tumors (brown nodes) of bone as follows:

> These giant-cell tumors (including the giant-cell epulis) are still a highly controversial subject. Three questions are still widely discussed. The first concerns the nature of these nodes, which are regarded as true tumors by some authors, whereas others look upon them as an excessive regenerative tissue growth. The second question is concerned with the identity of

16. Coley, W. B., Prognosis in Giant Cell Sarcoma of Long Bones. *Ann. Surg.* 79:321 (March) 1924.
17. Weinmann, J. P., and Sicher, Harry, *Bone and Bones.* St. Louis: C. V. Mosby Company, 1947.

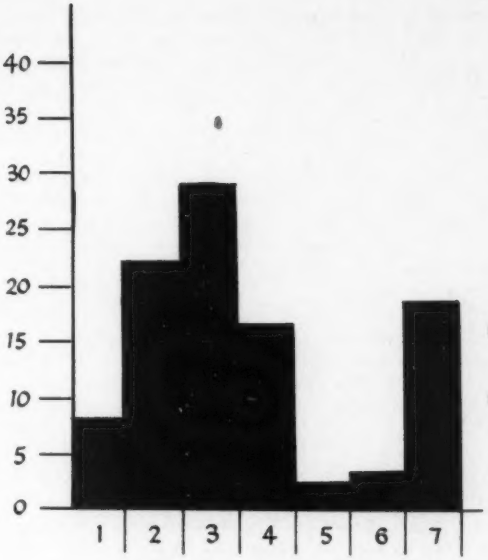


Fig. 2.—Duration of symptoms in giant cell tumors studied

the brown nodes in Recklinghausen's disease, solitary brown nodes, and the giant-cell epulis. The last and the more general question concerns the derivation and the biologic significance of the giant cells in the nodes.

Histology of the Giant-Cell Nodes.—The giant-cell nodes consist of a stroma of thin collagenous and mostly precollagenous or argyrophil fibers. The cells are very numerous and most of them are round or spindle shaped and could be classified as young fibroblasts and undifferentiated mesenchymal cells, according to Maximow's terminology. The giant cells, multinucleated cells of varying size and shape, are found more or less evenly distributed in the node. Their number in a unit area is highly variable. Differences between these cells and osteoclasts are slight. The localization of the nuclei in the center of the cell has been mentioned repeatedly as characteristic for the giant cells of the nodes. The changes in position of the nuclei from the periphery of the cell in the osteoclasts to the center of the free giant cells is, in all probability, only a sign of the inactivity of the latter.

Of great importance is the rich vascularization of the giant-cell nodes. The capillaries are especially wide, and the development of new capillaries by proliferation of the endothelium can be observed easily. Extravasation of red blood cells into the stroma is frequent and, in a brown node, it is almost the rule to find areas of fresh hemorrhage or an accumulation of hemosiderin pigment as a residue of previous bleeding. It is the frequency of these hemorrhages which is responsible for the reddish brown color of the nodes and for the widely used term brown tumors.

Brown nodes develop in the spongy bone of the epiphyses or in flat bones. Sometimes, in hyperparathyroidism, they arise in the spongy bone which had replaced the cortical layer of a bone. Their growth inside a bone, of course, leads to destruction of bone. The reaction of the bone to the development of the brown node is characteristic. To compensate for the slow resorption of bone from within, new bone is deposited on the periosteal, or outer surface, so that, for some time, destruction of bone and formation of bone are almost balanced. This

leads to a "swelling" or "thickening" of the involved bone, terms which should not be used without clearly stating the mechanism of the bone changes. The formation of new bone is seen by most authors as a walling in of the tumor or as a formation of a barrier against the progress of the tumor. This view is certainly erroneous. It stems from an analogy between a protective capsule of connective tissue and a bony shell. This analogy is not justified because connective tissue does offer resistance to pressure, whereas bone is removed by resorption even under slight pressure if the force acts in an unphysiologic direction. It seems to be much more in accordance with the biology of bone to view this phenomenon of formation of bone as a response to mechanical stimuli. It is the attempt of the organism to restore the strength of the involved bone which is weakened by the resorptive process. In extreme cases the bony shell may disappear in some areas of the node. A further growth of the node is then sometimes observed, being directed into the interstices between muscles, tendons, and ligaments but never infiltrating these organs.

A bony shell developing in the way just described is lacking only in one special type of giant-cell node, the giant-cell epulis. This growth originates in the bone of the alveolar process and is directed toward the surface. The bone covering the primordium of a giant-cell epulis is so thin that the process of resorption leads to total destruction of the plate before there is time for any compensatory apposition on the outer surface.

* * *

The origin of the giant cells in brown nodes is still highly controversial. There is not only the general question as to whether the giant cells differentiate from cells of the connective tissue or from endothelial cells, but also the specific question as to whether these giant cells are osteoclasts persisting after the destruction of bone or whether they differentiate in the tissue of the node without relation to the bone.

As far as the origin of the osteoclasts is concerned, it can be stated that the evidence points more and more away from an endothelial differentiation of the osteoclastic giant cells. The second question can be answered only tentatively as long as the evidence is equivocal. Although rejected by some authors, the assumption that giant cells of the brown nodes are persisting osteoclasts still seems to be justified. The arguments against it point out mainly slight differences in shape, staining reaction, and position of the nuclei in the nodular giant cells and osteoclasts. These differences could easily be explained by the fact that the typical osteoclasts are active bone-resorbing cells, whereas most giant cells in the node are in an inactive state.

The question must be answered as to whether it is probable that osteoclasts persist beyond the period of their resorptive activity. Normally, oseoclasts disappear in a short time after their task is completed. In fact, they disappear so quickly that it is almost impossible to learn anything about the mode of their disappearance.

Relation to Parathyroid Activity

Since 1930 there has been a rapid accumulation of information on parathyroid overactivity and its relation to certain types of bone pathology. The multiple bone cysts and solid masses within bone defects found in association with hyperparathyroidism at times show histologic findings identical with cysts and giant cell tumors. Some observers have concluded that all giant cell tumors and bone cysts are manifestations of, or results of, abnormal parathyroid activity. This sweeping deduction has met with considerable opposition, and it seems as yet unwarranted to classify these tumors as such. However, it seems essential, particularly in atypical giant cell tumors and especially around the jaws, that there should be a careful investigation for possible parathyroid disturbance.—*Department of Oral Histopathology, School of Dentistry, University of Southern California.*

UMI

TANTALUM IN CRANIOPLASTY AND ORAL SURGERY: FABRICATION TECHNIC AND REPORT OF CASE

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St. Albans, L. I., N. Y.

THE PURPOSE of this paper on tantalum is to acquaint interested members of the dental profession with its history, properties and possibilities as related to oral surgery, and to present a simplified practical technic for the fabrication of tantalum plates for the restoration of skull defects. In World War II tantalum stimulated the imaginations of the surgical profession because of the multiplicity of its indications for war injuries, particularly from the viewpoint of restorations of bony losses. Its use ranks in importance with the other major advances in the military surgery of this war, such as the employment of blood plasma, the sulfonamides and the antibiotics.

Literature

A review of the literature reveals that tantalum first was used for surgery in 1936 by Burch and Carney,[1] who employed it successfully in the form of bone plates, screws and wire in both animals and human beings. Prior to the recent war the unique properties and advantages of this singular metal for use in surgery did not seem to be appreciated fully. However, these properties were recognized by the military surgeon during the war, and today the advances in the knowledge of its application have been so rapid as to give rise to considerable enthusiasm.

At this point the historical background will be reviewed with reference to some of the chemical and physical properties of tantalum. In 1801 Hatchett,[2] while working on a strange black mineral, noticed the presence of a new element which he named columbium because the mineral had been found in North America. Anders Gustaf Ekeberg, a chemistry instructor at Uppsala College, Sweden, found a second element in the same material in 1803. Ekeberg spent his life trying to isolate it from its compounds. Because this search proved so tantalizing to him he named this elusive element tantalum, which is number 73 in the periodic table, after Tantalus, the ill-fated son of the mythologic Greek god Zeus.

One hundred years passed before a fairly pure tantalum was separated in metallic form. This was accomplished in Germany by a Russian, Werner Van Bolton. He produced it in the form of tantalum wire which was used as filaments in electric light bulbs. Although a small amount of tantalum was produced in Germany from 1903 until the beginning of World War I and was used to a limited extent in dental and surgical instruments, the cost of the tantalum was prohibitive for any great advances

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The opinions or assertions contained herein are the private ones of the writers and not to be construed as official or reflecting the views of the Navy Department or the Naval Service at large (Art. 113(2) U. S. Navy Regulations).

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1. Pudenz, Robert H., The Repair of Cranial Defects with Tantalum. *J.A.M.A.* 121:478 (February 13) 1943.
2. Olson, Carl T., Tantalum; Glimpse of Its Surgical Future. *Indust. Med.* 13:738 (September) 1944.

in its use. It was not until 1922 that it was produced on a commercial scale. The man responsible for this was Clarence W. Blake[2] of the Fansteel Metallurgical Corporation of North Chicago, who developed a new method of producing this interesting metal.

Production of Tantalum

Tantalum is found at the present time in ten different countries, originating in the form of iron and manganese ores and always associated with columbium. It requires enormous amounts of material to produce appreciable quantities of high-grade ore. For example, in the Black Hills of South Dakota approximately 2½ tons of rock must be mined, milled, concentrated and processed to produce 1 pound of tantalum. Processing the tantalum ore to produce pure tantalum is a combination of powder metallurgy, electrolysis and a process known as sintering. Pure tantalum is a bluish white metal with a density of 16.6 which makes it weigh twice as much as steel. It can only be cold-worked and becomes brittle owing to molecular stresses and strains set up by working. It can be annealed only in vacuum. By special working, tantalum may have a Brinell hardness ranging from 150 to 600. It has a thermal conductivity similar to steel and a coefficient of expansion about three tenths that of silver.

Properties

It is the chemical properties which make tantalum so valuable to the surgical pro-

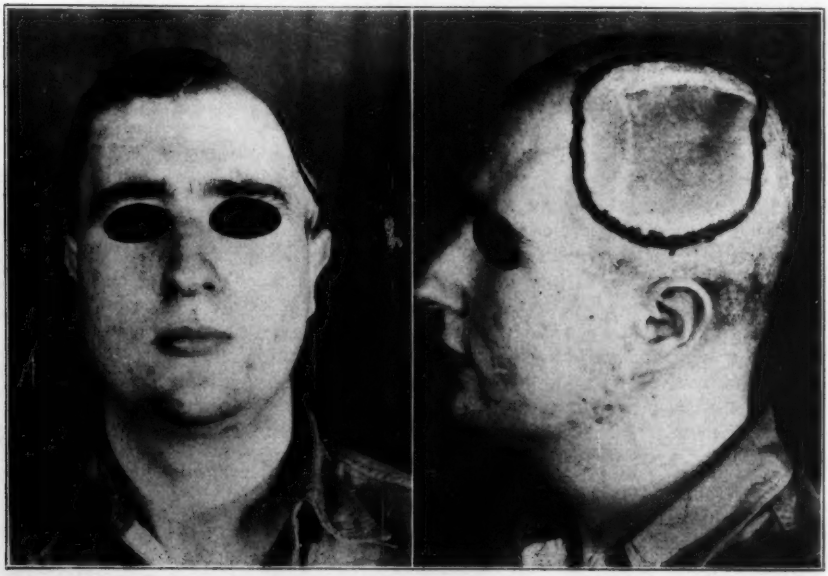

Fig. 1.—Left: Facial aspect showing extent of cranial deformity involving frontal region. Right: Lateral aspect showing indelible outline of cranial deformity, mainly of parietal region. Outline is 1 cm. beyond free border

Fig. 2.—Left: Stone model with 1 mm. groove over indelible outline rendering a permanent registration of extent of cranial deformity. Center: Same model held at angulation to illustrate lack of cranial contour. Right: Another aspect illustrating the groove 1 cm. beyond the border of the deformity

fession. Bothe and Davenport[3] in their work made the following conclusions: "Bone reactions were not closely correlated with the magnitude of potential differences, but remain characteristic for a given metal or alloy. Electrolysis is not the primary cause of unfavorable bone reaction. The primary causes are determined by the physical and chemical properties of the metal itself. Solubility and the degree of toxicity of the dissolution products appear to be the chief factors."

With these facts in mind let us consider the chemical properties of tantalum. So far as has been shown, no organic acid, salt, gas, alcohol, ketone, aldehyde or ester has any effect on tantalum. There are some exceptions such as reagents which contain fluorine, free sulfate or strong alkalis. The only acids which affect tantalum are hydrofluoric, fuming sulphuric and phosphoric, the latter two only at high concentrations and temperatures. To the oral surgeon, the neurosurgeon and the orthopedic surgeon this means that tantalum is a material available for their use which is completely inert to any chemical that may be produced in the body.

It will be interesting at this point to examine the work and opinions of some men who have done extensive work with this metal. In the field of neurosurgery tantalum is used in various forms—plate, foil, wire and ribbon. Utilizing these different forms it has been possible for the neurosurgeon successfully to repair cranial defects, secure hemostasis, perform accurate nerve suturing and prevent postoperative adhesions in brain tissues and the spinal canal after surgery. The utilization of the tantalum foil subdurally in head injuries prevents the formation of a meningocerebral cicatrix so often responsible for serious sequelae such as epilepsy and other neurogenic disorders.

Uses and Adaptability

The greatest use to which tantalum has been put during World War II and subsequent to that period is the repair of skull defects. In their report on the use of vitallium for the repair of cranial defects, Peyton and Hall state that an ideal cranioplastic material should be (1) nonirritating both electrically and chemically, (2) strong and inelastic, (3) light in weight, (4) nonabsorbable and (5) malleable. Tantalum fulfills all of these requirements except that of lightness in weight; it already has been stated that tantalum has a density of 16.6, or about twice the weight of steel. However, the adaptability of thin sheets of the metal compensates for this disadvantage. The fabrication of the tantalum plate to restore the skull defect requires the services of a dentist, for by virtue of his profession he is in almost daily

3. Bothe, R. T., and Davenport, H. A., Reaction of Bone to Metals. *Surg. Gynec. & Obst.* 74:231 (February 1) 1942.

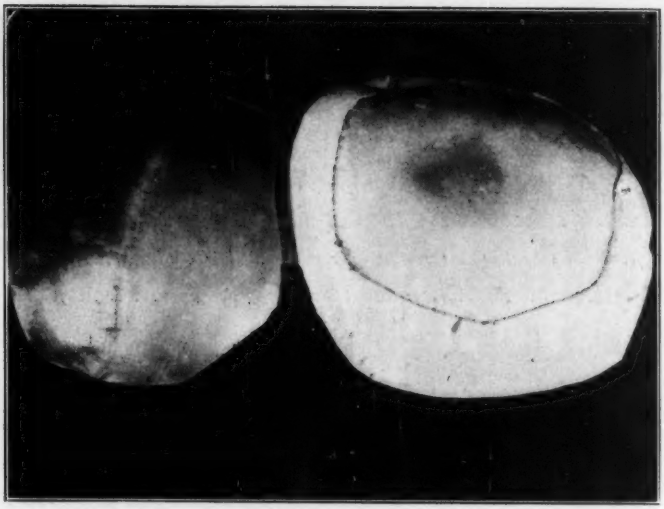

Fig. 3.—Corrected model with deformity filled in with wax, and wax pattern simulating size, curvature and contour of the contemplated plate

contact with the laboratory materials used and consequently has a decided advantage because of his understanding of the physical properties, manipulation and laboratory procedures involved.

Tantalum surgical sheets are available in varying thicknesses—0.0125, 0.0150, 0.020 inch; dimensions, 6 by 6 inches. Should larger sheets be required they are readily obtainable. For most cases requiring tantalum, the 0.020 inch thickness is used. It can be cold-worked successfully to the desired shape and, upon insertion, acts as an innocuous protective covering for the delicate cranial tissues and yet is able to sustain any ordinary external force.

Fabrication Technic for Tantalum Plate

There are many technics that can be utilized in the fabrication of a successful tantalum plate. Diggs[4] employs a technic modified after the direct method by Holtzendorff. Gerry[5] has developed a procedure in which metallic dies are not required in the fabrication process. Literature is replete with many other equally successful methods, but for purposes of simplification, the technic to be presented is to be considered as *a* method, rather than *the* method.

The technic involved in the fabrication of a successful tantalum plate does not require elaborate equipment. The physical requirements, with slight modifications, can be found in any dental laboratory. Basically, these are the essential materials used in the fabrication process: hydrocolloid, artificial stone, plaster, wax, asbestos strips, printer's type metal and, of course, tantalum sheet.

The patient's head should be close shaven. In consultation with the neurosurgeon

4. Diggs, John S., Direct Method of Forming Tantalum for Repair of Skull Defects. *J.A.D.A.* 34:745 (June 1) 1947.

5. Gerry, Roger G., A Simplified Technique for the Fabrication of Tantalum Skull Plates. *U. S. Nav. Med. Bull.* 46:1499 (October) 1946.

and a careful evaluation of appropriate roentgenograms, the prospective cranioplasty is taken into consideration. The cranial deformity is outlined with indelible pencil, with an allowance of 1 cm. beyond the border (Fig. 1, left and right). The extent of the impression must be determined. This is particularly true when the deformity involves the frontal bone or supra-orbital ridge. In these cases, the impression should include the entire frontal region, both eyes and, if deemed necessary, both sides of the temporal region. The purpose is obvious: to use the normal side as a comparative guide in reconstructing a similar bilateral contour. For the most part, cranial deformities of the temporal, parietal and occipital regions require a direct impression technic, since the contouring can be visualized by direct continuity of adjacent bone.

The dental chair should be adjusted so that the patient is comfortable and the cranial defect accessible. It is imperative that a horizontal impression field be established and maintained while the impression is being taken.

Hydrocolloidal materials are chosen because of their ease of manipulation, tissue tolerance and method of application. Should the extent of the impression include the orbital region, the eyebrows and eyelashes should be coated with vaseline. Otherwise they would become incorporated with the hydrocolloid. Finally, the impression should extend at least 2 inches beyond the indelible outline of the defect.

The hydrocolloidal material, sufficiently cooled, is painted on by means of a brush. After an approximate thickness of ¼ inch has been applied, two or three layers of dental gauze napkins, impregnated with hydrocolloid, are added. This is done to enmesh and keep intact the underlying basic impression material. After the hydrocolloid has set, a plaster mix is added, extending slightly beyond the hydrocolloid, and of adequate thickness to insure removal of the entire mass without distortion. Upon removal, the indelible outline should be demarked clearly in the impression. Once a satisfactory impression has been obtained, it should be matrixed and poured in artificial stone. After setting and subsequent separation, an original stone model with an indelible outline is obtained.

Fig. 4.—Left: Patient gently holding wax pattern in position. Right: Anterior view

Obtaining Satisfactory Contour

The next consideration is to correct the deformity. First, the indelible pencil outline is grooved approximately 1 mm. (Fig. 2, left, center and right). This renders a permanent recording of the outline. Filling in and contouring is accomplished by the use of ordinary pink wax. When the reconstructed contour is satisfactory, the patient is recalled for a trial fit. A single thickness of pink wax is adapted over the model, thus obtaining a corrected pattern, simulating the size, curvatures and contour of the contemplated plate (Fig. 3). By placing the chilled wax pattern in position on the patient, a direct means is obtained for minor corrections (Fig. 4, left and right).

When satisfactory, the corrected model is ready for reproduction as a metallic die. Three or four triangular grooves are cut along the free border of the model. It is vaselined and inverted into quick setting plaster, with either wax or cardboard as a suitable matrix. At least ½ inch thickness of plaster in all dimensions is necessary for obtaining a satisfactory mold. The recovery of the corrected model is accomplished by removing the matrix and carefully cleaving. Usually, a clean midbreak will dislodge the model. The plaster segments then are reassembled and secured in position (Fig. 5, left). When the mold is dry, it is poured in printer's type metal. Its characteristic expansion upon solidifying makes printer's type metal ideal for dies.

After obtaining a satisfactory positive die (Fig. 5, right), a counterdie is the next consideration. The grooved outline on the positive die is remarked with an indelible pencil. By adapting moist asbestos strips and trimming within the indelible markings, an asbestos layer is obtained. This is glued into position. Then a matrix lined with asbestos strips and re-enforced with plaster is made. A heat resisting separating medium, fire clay or talc in alcohol, is applied to the exposed borders to prevent fusion. The counterdie is poured, permitted to cool and subsequently separated. The result is an interlocking die and counterdie.

The proper cutting of the tantalum sheet is accomplished by utilizing a thin cardboard templet. Then the tantalum sheet is interproximated between the dies and, depending on the size and curvatures, is pressed slowly in position. Final closure, in a flask press, is maintained for twenty-four hours, after ascertaining that the interlocked dies are in contact at all their peripheries.

Upon separation, the tantalum plate should retain its new shape and, when placed on the original model, should conform with the marked outline.

Finishing the plate entails multiple perforations, not only to receive tantalum screws but also to permit drainage. Specified holes at the border are made by using No. 702 SHP burs, followed by No. 40 drills, and finally countersinking them with spear point vulcanite burs to such a depth as to accommodate the head of a tantalum screw. Drainage perforations are dispersed over the entire plate, approximately ½ inch apart. These need not be countersunk (Fig. 6).

The plate should be smooth and without metallic irregularities. All rough edges are removed with stones and excessive scratches reduced with coarse pumice. The plate is repressed for two hours to correct any changes caused by drilling and polishing.

Operation

Absolute cleanliness is imperative. The plate should be washed in a grease solvent, carbon tetrachloride or xylol, rinsed and then pickled in concentrated sulfuric acid for at least ten minutes to eliminate all foreign impurities. It is then washed with

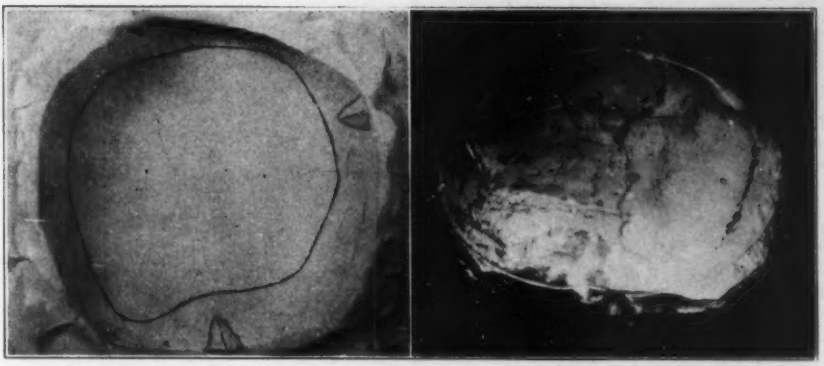

Fig. 5.—Left: Plaster mold reassembled. Lines indicate outline of groove and position of triangular grooves at the borders. Right: Positive die with all irregularities removed

soap and water, dried and forwarded for sterilization and insertion. Handling should be at a minimum. Fingerprints can be removed by means of alcohol.

The placing of the plate is done by a neurosurgical team (Fig. 7, left and right) and may be accomplished in various manners. The method most universally used is the inlay technic and the securing of the plate by tantalum screws. It is reported by Carl T. Olson, medical director of the Fansteel Metallurgical Corporation, that tantalum screws and tantalum plates have been used for more than seven years without any evidences of foreign body irritation, and that the regeneration of bone in the neighborhood of the tantalum is quite remarkable. There have been instances in laboratory animals where 3 inch defects in the skull have been covered completely over with bone following the insertion of a tantalum plate.

All the other branches of surgery have been busy utilizing this wonderful metal. Yet there is very little to be found in the literature of oral surgery about the use of tantalum in the treatment of bony defects and malunions of the jaws. Christiansen,[6] who did some early work with tantalum at the Naval Dental School, Bethesda, makes the following statement concerning the use of the metal in open reduction of jaw injuries: "Tantalum or stainless steel wire of 0.025 gage tightened by twisting after passage through holes drilled in these parts is, frequently enough, in extreme displacement when bone loss exists, and greater rigidity is required. For this, a tantalum plate secured by screws is well tolerated by both the bone and the soft tissues. This inert metal is valuable to bridge gaps where bone substance is missing and it has been used for this purpose to stabilize bony parts in the presence of infection where bone or cartilage inserts probably would be promptly cast off."

A number of cases of tantalum implants in jaw injuries were observed at the National Naval Medical Center, Bethesda, Md., and at no time did any of them cause undesirable tissue reaction. There were cases where the plates were removed, and the bone and soft tissues around and in contact with the metal were in the highest state of health. It was observed at Bethesda and also at the U.S. Naval Hospital, St. Albans,[7] that the presence of the metal seemed to accelerate bony

6. Christiansen, G. W., Open Operation and Tantalum Plate Insertion for Fracture of the Mandible. *J. Oral Surg.* 3:194 (July) 1945.

7. Maxwell, M. M.; Schork, C. J., and Heidt, Edward, Rationale of Treatment in Maxillofacial Injuries with Report of Four Cases. *J. Oral Surg.* 4:269 (October) 1946.

Fig. 6.—Tantalum plate showing multiple perforations for reception of screws and drainage. Die and counterdie show outline and position of interlocking triangles

regeneration. The uses in which such a metal in the form of plates, wires, screws, foil and tantalum wool can be used in oral surgery, are many; that is, in losses of bony substance, retention of a posterior segment, correction of malunion, open reduction of badly comminuted fractures, reduction of edentulous fractures of the jaws, restoration of facial contours and symmetry in disfiguring facial injuries and as an adjunct to the surgical closure of oromaxillary fistula. Bellinger,[8] in his preliminary report on the use of tantalum in maxillofacial and oral surgery, presented

8. Bellinger, Don H., Preliminary Report on the Use of Tantalum in Maxillofacial and Oral Surgery. *J. Oral Surg.* 5:108 (April) 1947.

Fig. 7.—Left: Esthetic result with tantalum plate in position. Right: Lateral aspect showing restoration of cranial contour

8 interesting cases in which tantalum has been utilized successfully. Tantalum is available to the oral surgeon and it seems advisable for him to take advantage of its unique properties to meet better the problems presented in every day practice.

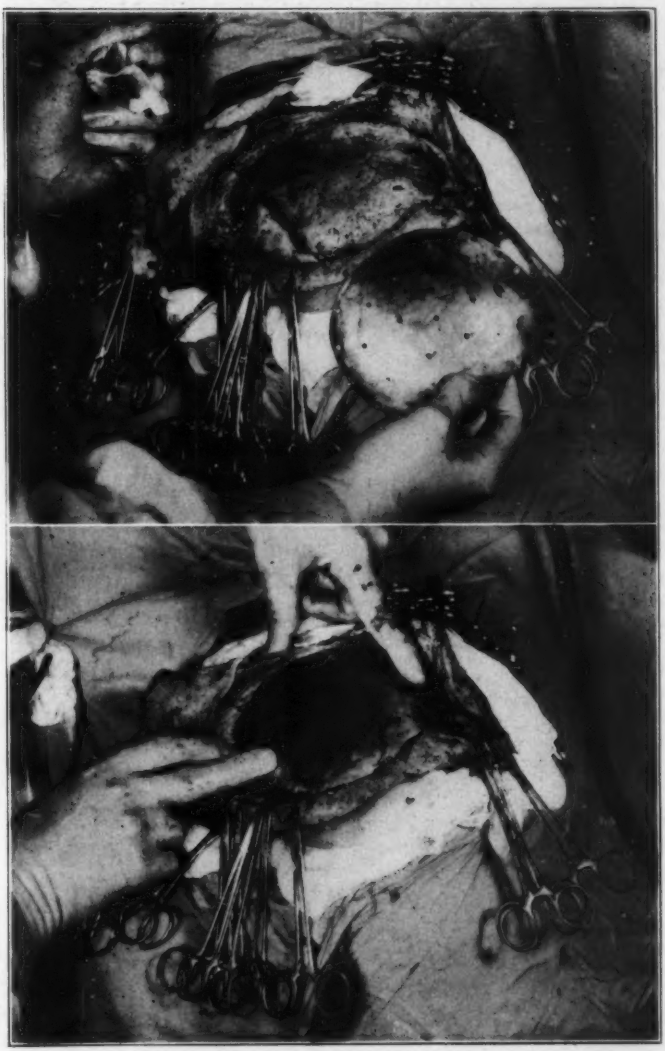

Fig. 8.—Above: Cranial defect exposed with plate beside it for comparison. The cortex of the brain was not visualized. Below: Tantalum plate in place showing good alignment to the external bony deficit

UMI

Report of Case

History.—A 20 year old white man first was admitted to the sick list from Boot Camp on June 30, 1945, at U. S. Naval Hospital, Sampson, N. Y., with the diagnosis of acute otitis media. He had a previous history of bilateral mastoidectomy in childhood but had had no further trouble until entering the service.

On July 3, 1945, he developed acute arthritis of the right ankle. A diagnosis of acute rheumatic fever was established, and on September 8, 1945, he was transferred to U. S. Navy Hospital, Palm Beach, Fla., to convalesce.

An attack of tonsillitis was treated with sulfonamides on November 7, 1945, and a tonsillectomy was performed on December 11, 1945. Nine days later he developed two convulsions. On the following day he complained of weakness in his right arm.

By December 26, 1945, he exhibited a right hemiplegia and aphasia. Streptococcus viridans was recovered from his spinal fluid. He was treated with enormous doses of penicillin and sulfadiazine.

Operation.—By December 30, 1945, he became comatose and was transferred to the U. S. Naval Hospital at Bethesda. A left frontoparietal craniotomy was performed and the rolandic vein was found thrombosed and infected. No localized abscess was evident by needling.

The patient improved slowly, but on January 25, 1946, following a localizing ventriculogram, a 6 cm. purulent abscess was removed entirely from the left premotor region. He was continued on chemotherapy and on April 3, 1946, a left parietal craniotomy was performed with resection of metastatic multiple abscesses. He was given penicillin intrathecally, intraventricularly and intramuscularly. Improvement was slow and steady, and by August 31, 1946, he was able to go on convalescent leave.

Cranioplasty.—On October 15, 1946, he was transferred to this activity for observation, rehabilitation and cranioplasty. At that time he presented an enormous skull defect on the left parietal region, a right hemiplegia and marked aphasia. He received physiotherapy and occupational therapy and showed marked improvement. On April 10, 1947, a cranioplasty was carried out, and a 13 by 13 cm. tantalum plate was inserted in the skull defect (Fig. 8, above and below).

Results.—Cosmetically, the results were good. His hemiplegia has improved markedly. He now gets about well with the aid of a leg brace. His speech is still impaired, but if he talks slowly he can make himself understood. He is completely controlled on anticonvulsive therapy to the extent of Dilantin Sodium (Parke, Davis & Company) grains 1½ and phenobarbital sodium grain ½ three times daily. He has been discharged from the service and is continuing his rehabilitation at home.

Mandibular Reconstruction in Jaw Deformities. Bernard N. Soderberg and John M. Mulvey, in *Plast. & Reconstr. Surg.* 2: May, 1947.

Summary

The principles of bone healing seen in primary fracture in general were repeated following bone transplantation. Cancellous bone was used as graft material because of its superior osteogenic properties. Most grafts were partially or completely decorticated to assist a more rapid vascularization. Little or no decortication was done in ridge grafts employed where some mechanical help in maintaining position of the edentulous fragment was thought helpful.

A balanced general metabolism with an adequate bone graft situated in a good soft tissue bed with an efficient minute circulation, is the ideal to be attained. Given this situation and proper fragment fixation maintained for a sufficient length of time, mandibular continuity becomes permanently established.

CONGENITAL BILATERAL HEMANGIO-ENDOTHELIOMA: CLINICOPATHOLOGIC REPORT

Malcolm Wallace Carr, D.D.S.,
New York

A MULTIPLICITY of pathologic conditions are encountered during the course of a routine clinical experience. Generally considered, the nature of disease is understood reasonably well and the treatment indicated is a matter of common knowledge. Occasionally, however, unusual, uncommon or even rare conditions are encountered about which little or nothing is known, notwithstanding the present advanced status of medical science. It is the case within this category that presents the most perplexing problems in diagnosis and causes the greatest concern in attempting to formulate some rational method of treatment. Indeed, it is not an uncommon experience in clinical practice to reach an impasse where it must be confessed that in a given case no known treatment is likely to be of any therapeutic value.

The author presents a report of a case of congenital bilateral hemangio-endothelioma of the face. The extraordinary appearance of the lesion placed it categorically in the group of anomalous conditions classified in clinical experience as unusual or rare. Review of the literature failed to reveal a published record of a case similar to it. The unusual nature of the lesion and the general interest manifested by all consultants who saw the patient make a published report desirable. Furthermore, it should be considered a professional obligation to publish, with complete clinical notes, reports of patients presenting unusual features of symptomatology or conditions of rare occurrence. Important data of value to clinical research frequently is lost to medical science because of certain uncontrollable factors which make it difficult or impractical to compile detailed notes on history or complete record of findings. The unfortunate inability to obtain permission for autopsy is one of the uncontrollable factors which many times make it impossible to render a complete report.

Although the record of the patient presented here may appear to be deficient in some details of investigation which appropriately might have been pursued during clinical study, the report is reasonably complete as to general history and physical examination, detailed description of the clinical appearance of the lesion and compilation of accurate notes on the course of the disease during the eight weeks it was under observation, from the seventh week of life until fatal termination at the age of 15 weeks. A detailed report of the postmortem findings is included as part of the record.

Report of Case

General History.—An infant girl, aged 7 weeks, born of Italian parents, was admitted to the surgical service of the Fifth Avenue Hospital, New York, on November 18, 1943. The chief complaint was swelling of both cheeks, present since birth, and ulceration of the lower lip since two weeks after birth.

The child was delivered at term by a midwife. She was a normal child in all respects, except for bilateral enlargement of the cheeks. The weight at birth was 9 pounds. The best weight was 10½ pounds at 5 weeks of age. There had been no previous illness, accident or operation. Both parents were living and well. There was one other child in the family, a normal

boy, 4 years of age. The past history and the family history otherwise were essentially unimportant.

The history of the present illness dated from birth. At birth both cheeks appeared slightly enlarged, the left more than the right, and the skin was marked by dilatation of the superficial vessels. Two weeks after birth a small ulcer occurred on the lower lip beneath the vermilion border and near the left commissure of the mouth. The child was seen at that time by a neighborhood physician who prescribed a colorless liquid preparation for local application. The use of the solution seemed to aggravate the ulcer and the lesion extended considerably after several applications.

Two weeks before admission the mother noticed two small nodular masses on either side of the neck just beneath the angle of the mandible. The swellings in the cheeks and the nodular masses in the neck had increased only slightly since the onset.

Two days before admission the child was seen by a physician at a welfare clinic who advised that she be admitted to a hospital for observation and diagnosis. Since birth the child's general health had been good. She had been bottle fed, had taken her feedings well and had gained some weight. The mother stated that the nipples had been boiled before using.

Physical Examination.—General physical examination at the time of admission revealed a well nourished, well developed child of 7 weeks of age, who was not acutely ill. The temperature, pulse rate and respiration rate were normal. Weight was 9 pounds 13 ounces.

There was a prominent swelling in both cheeks, the left cheek appearing larger than the right. The swelling on the left side extended from the zygomatic arch and malar bone to the lower border of the mandible. There was some diffuse extension below the lower border of the mandible into the tissues of the neck. The swelling also extended postero-anteriorly, from just anterior to the ear and the angle of the mandible to the commissure of the mouth. In the postero-anterior direction the swelling appeared to be separated into two portions so that the posterior portion appeared to be in the region overlying the masseteric fascia, whereas the anterior portion occupied the region commonly referred to as the cheek, anterior to the anterior border of the masseter and as far forward as the commissure of the mouth, the ala of the nose and limited above by the inferior border of the orbit. The anterior portion appeared more distended. The appearance of the right side of the face corresponded in a general way to the left side, except the swelling of the right side was less pronounced.

Telangiectasis was pronounced in both cheeks and extended into the tissues of the neck. There was a small encrusted ulcer 0.5 cm. in diameter in the center of the anterior portion of the swelling of the left cheek. The general appearance of the swelling was that of extreme distention with dilatation and engorgement of superficial terminal vessels. The skin was tense, shiny and red. There were several areas 1 cm. in diameter that appeared purplish or bluish in color. Upon palpation the mass was soft and compressible. It gave a distinct sense of fluctuation over a considerable region. It was not tender. Pressure applied to the swelling did not elicit pitting and caused no symptoms of subjective sensation. The swelling extended through the entire thickness of the cheek so as to bulge the buccal mucous membrane within the mouth and seemed to prevent the child from fully closing the mouth.

There was a large, deeply excavated, sloughing ulcer on the lower lip, 2 cm. in diameter. The center of the ulcer was situated to the left of the median line and occupied the greater portion of the left side of the lip extending through the vermilion border but not extending to the inner surface of the lip.

Intra-oral examination revealed several small superficial ulcers (aphthae) situated on the alveolus and in the buccal mucosa. There was no ulcer in the buccal mucosa on the left side corresponding to the position of the encrusted ulcer on the external cheek. The floor of the mouth was elevated although not inflamed. The tongue was pushed slightly back. Salivary secretion was normal or slightly hyperactive. Throat examination was negative. The submaxillary and submental lymph nodes were palpable on both sides.

Both ear canals were obstructed by narrowing from external pressure on the floor and posteriorly. The obstruction was soft so that an applicator could be passed easily. It was not possible to see the tympanic membranes. There was a small amount of purulent discharge from

both ears. Examination of the mastoid region was negative. There was no deformity, obstruction or discharge in the nose. Examination of the eye elicited normal reaction to light and accommodation. There was no strabismus, nystagmus, ptosis or exophthalmos. The physical examination otherwise was essentially negative for any abnormalities of function, growth or development.

Roentgenographic films taken of the skull and of the bones of the face in the lateral direction showed no evidence of bone disease.

Clinical Pathology.—A hematologic report showed: hemoglobin 70 per cent; red blood cells 4,300,000 per cubic millimeter; white blood cells 18,400 per cubic millimeter; polymorphonuclear leukocytes 61 per cent; lymphocytes 33 per cent; monocytes 3 per cent; eosinophils 3 per cent; platelets 240,000, and coagulation time four minutes.

Urinalysis revealed an acid reaction; a faint trace of albumin; absence of sugar, acetone and diacetic acid; few white blood cells, and the presence of amorphous urates.

A bacteriologic smear from the ulceration on the lip was negative for fusospirochetes. A culture from the throat was negative for diphtheria bacillus. A vaginal smear showed many gram-positive and gram-negative cocci to be present; no gram-negative intracellular diplococci, but occasional pus cells.

In serologic examination reaction to the Wassermann test was negative.

Differential Diagnosis.—Clinical examination revealed four pre-eminent concomitant features: bilateral soft swellings of the cheeks marked by dilatation of the superficial vessels; a large, deeply excavated ulcer of the lower lip and a small encrusted region of the left cheek; multiple small, superficial ulcers within the mouth, and moderate enlargement of the regional lymph nodes.

The ulceration of the lower lip and the small ulcer in the left cheek appeared to be the most active lesions, and seemed at first to present some of the clinical features of cancrum oris (noma). However, after more deliberate consideration the provisional diagnosis of noma was abandoned because the lesion appeared to have had its origin on the external skin surface with no deep ulceration in the mucosa of the cheek or lip at a position corresponding to the external ulcer. Noma is a rapidly progressive gangrene of the mouth usually extending by thrombosis caused by the invasion of the tissues by fusospirochetal or other organisms, and producing marked symptoms of toxemia, high temperature and rapid and weak pulse. The ulceration in this case was localized and showed no tendency to extend rapidly.

Cellulitis, as such, was not present, nor symptoms of general toxemia. Furthermore, the local application of some (caustic) drug before admission to the hospital, as recorded in the history, indicated that the lesion was primarily superficial with secondary invasion. There was no evidence of primary infection such as pustules. The primary cause of the original ulceration very likely may have been circulatory disturbance in the terminal branches of the superficial vessels at the median line caused by pressure by the tumor masses.

The diagnosis of telangiectasis of the cheeks was evident, but opinion varied as to the exact nature of the underlying tumor mass. The tumor had some of the clinical characteristics of a cyst of the parotid gland although an attempt to aspirate the left cheek was unsuccessful. Mixed tumors of the parotid gland and lipoma were considered, but the general appearance and texture did not favor this opinion. Mikulicz's disease was considered also, but in this condition there is a symmetrical enlargement of the salivary and lacrimal glands which is limited strictly to these glands and does not invade extracapsular structures. Furthermore there is a decrease in secretion and the principal symptom, accordingly, is dryness of the mouth. These factors were not present in the case under consideration.

Congenital elephantiasis with telangiectodes was considered, but the clinical appearance did not suggest an overgrowth of connective tissue; therefore the lesion did not have the characteristics of elephantiasis. Nevus vasculosus and lymphosarcoma also were suggested. The lesion appeared more lymphoid in nature and it was believed to be histologically a benign tumor composed of a combination of both lymphangiomatous and hemangiomatous elements.

The ulcers in the mouth were nonspecific, superficial aphthae. Negative bacteriologic ex-

amination for fusospirochetes and Oidium albicans ruled out fusospirochetal infections (Vincent's stomatitis) and thrush.

The clinical diagnosis was as follows: (a) benign neoplasm—congenital bilateral lymphangioma and hemangioma involving the entire cervicofacial region; (b) congenital bilateral telangiectasis; (c) ulceration of lower lip; primarily a chemical slough with secondary infection superimposed on congenital telangiectasis; (d) aphthous stomatitis; (e) submental and submaxillary lymphadenitis—secondary to ulcerations of lip and stomatitis.

Prognosis.—The tumor was considered inoperable because of its fundamental characteristics, diffuse nature and its wide bilateral extension over the entire cervicofacial region. Nor was it considered that radiation, either radium or roentgenotherapy, or any other form of treatment would be of any value. The prognosis was unfavorable. Biopsy was not done.

For a period of five weeks the condition extended gradually, continually invading the deeper tissues of the neck (Figs. 1 and 2). The sixth week of observation was notable because of a period of remission of acute symptoms accompanied by remarkable general improvement not attributable to any known cause. This one week period of remission was followed, however, by a period of recrudescence during which there was an acute exacerbation of all symptoms,

Fig. 1.—Photograph of left side of face taken three weeks before death. Well defined limitations of extreme distention and extensive telangiectasis are evident. Ulcers on lower lip and left cheek have healed while vermilion portion of lower lip has been destroyed by ulceration. Cicatricial contraction at angles of mouth, together with bilateral pressure caused by tumor mass and distention of floor of mouth, has caused lips to appear puckered and prevent child from bringing lips together in normal position

terminating fatally at the end of the eighth week of observation. The tumor mass had infiltrated tissue over an extensive region and developed to enormous proportions as shown at the postmortem examination. Microscopically the tumor was diagnosed as hemangio-endothelioma.

Autopsy.—Postmortem examination begun nine hours after death showed the body of a rather poorly nourished infant girl measuring 57 cm. in length and weighing 4,190 Gm. (approximately 9¼ pounds).

The lower part of the face and the neck were enlarged greatly and deformed in an almost symmetrical fashion. The cheeks appeared to be puffed out. The parotid and submaxillary regions were swollen markedly and slightly firm in consistency. The circumference of the head at the level of the lower and widest part of the face exceeded that of the thorax (Fig. 2). Swelling of the right parotid region extended as high as the level of the zygoma. On the left side swelling was more marked and extended up into the temporal region to a level 1 cm. above the ear.

Diameters of the head measured as follows: occipitofrontal 13 cm.; occipitomental 16 cm.; suboccipitobregmatic 11 cm.; biparietal 10.5 cm. The circumference of the head in the occipitomental diameter was 45 cm. and in the occipitofrontal diameter was 38.5 cm. The circumference of the enlarged neck was 17 cm. The anterior fontanel measured 3 by 2.5 cm. in the largest diameters. The pupils each measured 3 mm. The palpebral conjunctival mucous membranes were pale.

Superficial blood vessels of the cheeks were dilated and plainly showed through the skin. The skin over the chin was mottled by hemorrhagic spots. The mouth orifice was smaller than normal. The upper lip near the angles of the mouth was swollen slightly, and the corresponding mucosa was mottled reddish purple. The floor of the mouth was elevated and the tongue was retracted somewhat. The hard palate was high and rather narrow, and the pharynx was narrowed slightly. On the left side the firm swelling of the cheek extended anteriorly as far as the lips, and it extended inferiorly to involve the gingivae of the mandible.

The tumor and neck organs were removed en masse (Fig. 3). The tumor then was seen to be nearly symmetrical and to be made up of a central submental-sublingual-submaxillary mass measuring about 7.5 by 4 by 2.5 cm. and of two attached lateral masses from the parotid and cheek regions, the left measuring about 9 by 4.5 by 2.8 cm. and the right about 7.5 by 4.5 by 2.5 cm.

Grossly the tumor was rather uniform in character, being reddish purple and fairly firm. Cut surfaces were purplish red and finely granular with mottlings of gray in places. There was a tendency toward formation of irregular lobules several millimeters in diameter. In places a thin fibrous capsule was visible, but in others a capsule was missing, and there was no sharp boundary between tumor and adjacent tissue. From the right parotid mass, tumor extended into the buccal fat pad in the form of small, connected, rounded nodules. Normal parotid and submaxillary gland tissue could not be identified. Tumor was not adherent to the skin. In the midline it was attached rather firmly to the periosteum of the inferior and posterior surfaces of the mandible. More laterally it was adherent to the anterior, inferior and internal surfaces of the mandible. A considerable amount of tumor lay between the mandible and pharynx.

Dissection of the neck organs revealed extension of the submental tumor mass downward in the midline anteriorly over the trachea and thyroid nearly to the level of the aortic arch. This cervical mass measured approximately 3.5 by 2.5 by 1 cm. in greatest diameters. The surface of this tumor was for the most part smooth, but in places adipose tissue was attached rather firmly and a capsule could not be recognized.

In close association with this tumor a number of 2 to 10 mm. sized, similar rounded nodules were attached anteriorly to the thyroid capsule, and a similar nodule measuring 12 by 8 by 3 mm. was attached to the capsule of the left side of the thymus. Cut surfaces of these nodules were pinkish red and glistening, resembling the main tumor.

The left superior, deep cervical lymph nodes were enlarged, and a half dozen of these closely approximated nodes formed a mass measuring 2.5 by 1.2 by 0.7 cm. in greatest diam-

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Fig. 2.—Photograph taken three weeks before death, showing bilateral extension into neck. Enormous growth and distention of tissue is evident. Width of tumor mass actually exceeded width of thorax

eters. A left retropharyngeal lymph node measured 10 by 7 by 3 mm. Some of these lymph nodes were rather soft in consistency, and when sectioned presented pinkish gray, velvety cut surfaces.

The external carotid arteries were larger than the internal carotid arteries. The left external carotid artery immediately divided into one large branch (this being equal in size to the internal carotid artery) and two smaller branches, the large branch being distributed to tumor in the parotid region, and the two smaller to tumor in the submaxillary region. The right external carotid artery, after giving off two rather small branches, entered the tumor of the parotid region with a diameter equal to that of the right internal carotid artery. The left internal carotid artery at its point of origin made a 90 to 120 degree turn downward, then coursed posteriorly and laterally around tumor to continue upward at reduced diameter between pharynx and tumor. The right internal carotid artery just beyond the point of origin made a turn of 80 degrees in its course around the border of the tumor.

The thorax was shaped normally. The umbilicus lay a little below the level of the costal margin. Axillary, epitrochlear and inguinal lymph nodes were not palpable. The skin was slightly loose on the upper extremities. In the subcutaneous abdominal tissue and omentum there was very little fat. The abdominal, pleural and pericardial cavities contained no excess fluid, and the surfaces were smooth and glistening.

The heart was enlarged moderately and nearly filled the pericardial cavity. It weighed 42 Gm. and was hypertrophied generally. The superior vena cava was dilated to a diameter of 11 mm., being a little larger than the thoracic portion of the inferior vena cava. The right auricle of the heart was dilated markedly, and the auricle wall in places was as thin as paper. The right ventricle was dilated moderately. All heart chambers contained thin, jelly-like clot and a little liquid blood. The foramen ovale was open as a narrow slit. The ductus arteriosus was closed. The myocardium was moderately firm in consistency. The valve leaflets looked normal. Valve ring circumferences measured as follows: tricuspid 5.5 cm.; pulmonary 3 cm.,

mitral 4 cm., and aortic 2.6 cm. The right ventricle wall measured 1.5 to 3 mm. in thickness, and the left ventricle wall measured 8 mm. in greatest thickness.

The lungs were voluminous and did not sink back when the thorax was opened. The left lung weighed 48 Gm. and the right weighed 63 Gm. Pleural surfaces were pinkish red with some purplish red mottlings visible posteriorly. Consistency was increased somewhat generally, and crepitation was not obtained readily. The large bronchi contained some foamy and mucoid blood-tinged fluid, and the mucosa was hyperemic. Bronchial lymph nodes were small. The cut surfaces of the lungs were redder than normal. A small amount of foamy fluid could be expressed from the cut surfaces of the left lung, and a moderate amount was expressable from the cut surfaces of all three lobes of the right lung. The posterior part of the right upper lobe showed a small, slightly firm, poorly outlined, dark red region.

The peritoneal surfaces of the intestines were pinkish gray and smooth. The cecum was mobile on a long mesentery. The sigmoid colon was longer than average or normal. It measured 25 cm. in length and at the point of greatest distention had a diameter of 3 cm. Except in the sigmoid colon, the intestines contained little fecal material. The intestinal mucosa was generally a little redder than normal. Peyer's patches were not large, and the lymphoid follicles of the colon were barely visible.

In the upper part of the ascending colon over a length of 2 to 3 cm. the intestinal wall was thickened to measure 3 mm. and was a little stiffer than normal. The mucosal folds here were flattened out and thickened, and the mucosa surface was grayish red. There was no narrowing of the lumen. The ileocolic lymph nodes were fairly large. Lymph nodes in the mesentery of the small intestine were small and numerous. Gross sections of these nodes looked normal. The stomach contained about 3 cc. of greenish brown turbid fluid and mucus. The mucosal surface was light pinkish red and smooth.

The spleen was enlarged somewhat, weighing 18 Gm. and measuring 5.5 by 3.5 by 1.5 cm. in greatest diameters. Externally it was purplish red in color. Consistency was normal. The cut surface had a deep pinkish red color. The pulp remained at the level of the cut capsule and could not be scraped off readily on the edge of the knife. Gray nodules representing Malpighian bodies were small and inconspicuous.

The liver was slightly enlarged, weighing 225 Gm. and measuring 13 by 8.5 by 4 cm. in greatest diameters. The surface was smooth and had a light brownish red color. Edges were sharp. In the cut sections nutmeg-like markings were visible in a few regions. The pancreas was not remarkable except for the fact that the cut surfaces showed a distinct tint of pink mixed with the usual gray color. The suprarenal glands together weighed 6 Gm. In cut sections the cortex was bright yellow. The brown inner zone showed no abnormal softening.

The kidneys together weighed 44 Gm. External surfaces and sectioned cortex were grayish red. The medulla was darker red. Architectural markings were normal. The urinary bladder contained about 20 cc. of urine. Mucosa was pinker than usual.

The thymus was normal in size and weighed 13 Gm. It extended down over the pericardial sac to the level of the fourth rib. The color externally was grayish pink, and the consistency was soft. The cut surface was reddish gray and glistening. The thyroid was normal in size, firm in consistency, and on section showed a light brown, finely granular surface.

The trachea, aorta and esophagus and the retroperitoneal lymph nodes were not remarkable. There were no gross deformities of the long bones. The line of ossification of a rib was straight, and the bone marrow was dark pinkish red. The meninges looked normal.

The brain weighed 600 Gm. and was not remarkable grossly except for moderate dilatation of the surface veins. After hardening in formalin solution numerous sections were made and no abnormalities were noted. The hypophysis looked normal.

Microscopic Examination.—Tissue was fixed in Zenker-formol solution, in 10 per cent formalin and in Kaiserling solution. Most of the sections were made from Zenker-formol fixed material and strained with hematoxylin and eosin. Sections of the neck and colon tumor also were stained with Masson and van Gieson stains. A number of blocks of the neck and face tumor were cut after fixation en masse in Kaiserling solution.

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Tumor.—Sections of ten blocks from various regions showed the tumor to be composed for the most part of atypical blood capillaries. In many regions, spaces of various sizes contained blood cells and each space was surrounded by a single layer of elongated cells, reproducing the picture of a blood capillary lined by normal endothelium. These capillaries were separated slightly from one another by loosely woven fibrous connective tissue. In less frequent regions blood-containing, endothelium-lined spaces were irregular, large and even cavernous in size.

Many parts of the tumor were more cellular than the areas just described. The capillary spaces were very close together, and the cells of the walls were larger, more irregular and more deeply staining than normal endothelial cells. Some small, red blood cell-containing spaces were separated from one another by only a single layer of cells, so that one cell was able to border part of the lumen of each of two capillary spaces.

There were also small spaces separated from one another by several layers of cells which formed a syncytium with, and were similar to, the cells directly bordering the spaces. No supporting connective tissue was visible here with the stains used. The cells of these areas were elongated or irregular in shape and were outlined poorly. Nuclei were vesicular, oval or irregularly rounded. Chromatin was usually in finely granular form, and a small nucleolus could be identified sometimes. Some nuclei were irregular in shape and stained deeply. A few mitotic figures were found after considerable search in the most cellular parts of the tumor. In the cellular regions, the smallest spaces often contained no blood cells. Parts of the tumor were traversed irregularly by fibrous tissue trabeculae which carried rather large arteries and veins.

Sections taken through the edge of the tumor showed no capsule. On the contrary, blood capillaries, atypical blood capillaries and more cellular masses of tumor cells were seen extending into adjacent striated muscle, adipose tissue and salivary glands in an invasive fashion. Striated muscle partly surrounded by tumor cells exhibited atrophy and partial necrosis. Muscles of the tongue were invaded extensively. Submaxillary and sublingual glands in places were invaded so extensively by tumor cells that few acini were recognizable. The parotid glands were invaded similarly but less extensively.

In a section through the lateral wall of the pharynx and the stylohyoid ligament, the muscles, connective tissue and mucosa were invaded by tumor tissue. Capillary spaces here were quite small and atypical. Judging from the large number of very dark staining nuclei and a few identified mitotic figures, the tumor was growing more rapidly here than elsewhere. In the various sections numerous nerves and lymphoid follicles were found surrounded by tumor, but the lymphatics contained no tumor cells.

Cervical Lymph Nodes.—In a section of a left deep superior cervical lymph node which measured 1.3 cm. in greatest diameter and was situated near the tumor, blood capillaries were found to be dilated greatly. Some polymorphonuclear leukocytes and red blood cells were found in the sinuses, and red blood cells were found also in phagocytic endothelial cells of the sinus walls. The lymphoid follicles were rather large and the germinal center cells exhibited degenerative changes. Sections of several other lymph nodes presented a similar picture with dilatation of blood capillaries prominent in all. Endothelial cells of the sinus walls of two of the lymph nodes contained considerable brown pigment, evidently derived from disintegrated red blood cells. No tumor tissue was identified in any of the nodes.

Colon.—Four sections from the thickened region in the ascending colon showed in the submucosa and mucosa large adjacent masses of closely approximated blood capillaries of the same type as those found in the less cellular parts of the neck and face tumor. The capillaries of the colon tumor were small, rather uniform in size and had walls composed of elongated cells with vesicular nuclei showing chromatin distributed in finely granular form. There were few dark staining nuclei, and mitotic figures were not found. Glands here were less numerous than normal, and some atrophic glands were separated widely by masses of atypical blood capillaries. Lymphoid follicles were surrounded by tumor. Muscle layers were not invaded or apparently distorted. Two sections of other parts of the colon, a section of stomach, and

a section of mesenteric lymph node showed slight engorgement of small blood vessels as the only significant change.

Lungs.—In sections taken from the upper and lower lobe of each lung, the alveolar wall capillaries were engorged moderately with blood, and one-half to one-third of the alveoli and some bronchi were filled with pink staining homogeneous material representing fluid. In the right upper lobe many alveoli contained a few polymorphonuclear leukocytes in addition to the indicated fluid. The wall of a small bronchus largely was destroyed, and the lumen region was filled with polymorphonuclear leukocytes. Another small bronchus showed less extensive but similar changes.

Other Organs.—The liver sinuses were distended somewhat with blood. About the efferent veins where engorgement was greatest, liver cells were shrunken and poorly preserved. The sinuses of the spleen contained an abnormally large number of blood cells, and some of the phagocytic cells contained greenish brown pigment. The blood capillaries of the heart, pancreas, suprarenal gland, kidney, leptomeninges and choroid plexus sections were overdistended with blood. The thymus appeared normal. In a rib the line of ossification looked normal and blood formation was very active. In sections of the brain Nissl's stain exhibited degenerative changes in numerous ganglion cells of cerebral cortex, basal nuclei, cerebellum, pons and medulla. Because some well preserved ganglion cells were also present, it appeared that the changes were not all caused by postmortem alteration. Neuronophagia showed a few ganglion cells in the pons. In the medulla of one suprarenal gland, there was a small mass of tumor cells having the histologic characteristics of a neuroblastoma.

Bacteriologic Examination.—No bacterial growth developed in a culture of heart blood in beef infusion broth.

Anatomic Diagnosis.—Large hemangio-endothelioma of face and neck with massive involvement of salivary glands and partial obstruction of pharynx and internal carotid arteries.

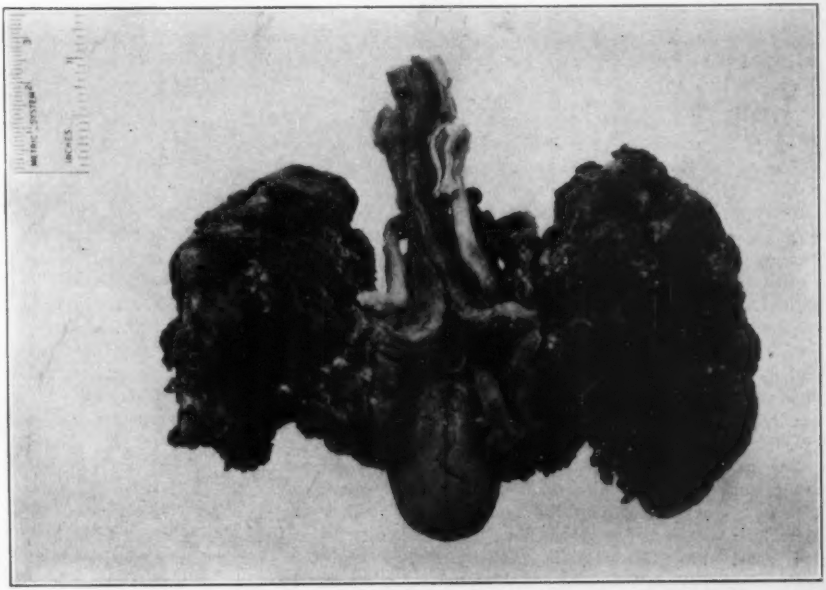

Fig. 3.—Appearance of tumor and floor of mouth, including tongue and neck organs, after removal en masse

Hyperplasia and chronic lymphadenitis of cervical lymph nodes. Hypertrophy and dilatation of heart. Passive congestion of viscera. Slight degenerative changes in ganglion cells of brain. Edema of lungs. Early lobular pneumonia of upper lobe of right lung. Hemangioma of ascending colon. Small neuroblastoma of suprarenal gland.

Summary and Conclusion

An unusual case of congenital bilateral hemangio-endothelioma with telangiectasis, involving the entire cervicofacial region in an infant girl, 7 weeks of age, is presented. The condition was under observation for eight weeks. During this period of observation and study accurate notes were recorded and are published in detail with this report. There was a period of remission of acute symptoms accompanied by remarkable general improvement not attributable to any known cause two weeks before death, which was followed by a period of recrudescence during which there was an acute exacerbation of all symptoms and fatal termination. The tumor mass infiltrated tissue over an extensive region and developed to enormous proportions causing only local symptoms of pressure and distention.—*52 East Sixty-First Street.*

Cavernous Sinus Thrombophlebitis. A Report on Six Consecutive Recoveries. J. L. Reid and F. McGuckin, Royal Victoria Infirmary, Newcastle-on-Tyne, England, in *J. Laryng. & Otol.* 61:273 (May) 1946.

A series of 6 cases recovering from cavernous sinus thrombophlebitis is presented. The cases illustrate the revolution in prognosis effected by modern methods.

The canons of diagnosis laid down by Eagleton twenty years ago are reviewed in the light of current experience. His criteria include: (1) the known site of infection; (2) blood stream infection; (3) early signs of venous obstruction; (4) involvement of nerves in the sinus; (5) neighborhood abscess of soft parts; (6) symptoms of complicating disease. In regard to these canons, the known site of infection may be nothing more than a trivial abrasion of skin. A negative culture from blood stream infection would not negate the diagnosis. Involvement of nerves in the sinus is open to criticism since fixity of the eye can result from edema and displacement. A reacting pupil is no bar to diagnosis. The neighborhood abscess of soft parts no longer holds good with prevention by modern therapy, and this is also true for meningeal and metastatic complications. If cavernous sinus involvement is considered as an extension of a process already begun in less vulnerable sites, the patient should be diagnosed on the gravity of his condition, increasing orbital edema, proptosis, chemosis, increasing fixity of the eye, thrombosed veins, local cyanosis, and the appearance of complications.

Successful therapy involves sulfonamide and penicillin treatment. Sulfamezathine taken orally is effective except in meningitis, where sulfadiazine is the drug of choice. A level of 8 to 10 mk. per 100 cc. of blood is maintained. Combined with this, penicillin is given intramuscularly, the dose being 15,000 units in 1 cc. of pyrogen-free solution every three hours. It is increased to 25,000 units in grave cases. Plasma and blood therapy are discretionary, and antigas gangrene serum is given when gas-infection is suspected. Nursing case is highly important. 2 references. 13 figures.—*Quart. Rev. Otorhinolaryng. & Br.-Esoph.* 6: June, 1947.

MANDIBULAR ASYMMETRY FOLLOWING OSTEOMYELITIS: REPORT OF CASE

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A GIRL, aged 23 years, had had osteomyelitis of the mandible on the left side at the age of 7 years. She had experienced the usual childhood diseases with no other serious sickness except for the osteomyelitis. She had had fourteen previous operations for this condition. The family history in relation to the disease was negative.

Examination.—There was marked asymmetry of the left side of the face with loss of normal angle contour causing considerable shortening of the mandible on the left side. There was a shifting of the midline to the left which was more pronounced on opening of the mouth. All teeth were missing on the left side up to the left cuspid (Figs. 1, left and center, and 2, left).

General physical findings were negative.

A laboratory examination of the urine showed it to be negative for sugar and negative for albumin.

Examination of the blood showed 3,800,000 erythrocytes per cubic millimeter; 8,000 leukocytes per cubic millimeter, and hemoglobin 90 per cent.

Treatment and Course.—On October 11, 1946, the patient was admitted to the hospital. After administration of thiopental sodium anesthesia, a diagonal osteotomy was performed on the left side intra-orally.

Previous to the operation, capped dental splints had been cemented to the remaining lower teeth and covered all of the upper teeth as well.

The osteotomy, starting at the alveolar crest in the third molar region, extended diagonally downward and forward to the cuspid region at the lower border of the mandible. The cutting of the bone was done by a series of drill holes extending through the entire thickness of the

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Fig. 1.—Photographs of the patient. Left: Before operation, showing open mouth position. Center: before operation, showing asymmetry with closed mouth. Right: After operation

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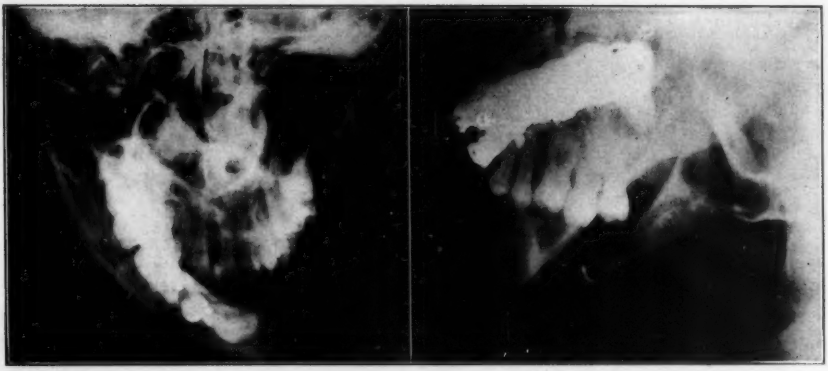

Fig. 2.—Roentgenograms. Left: Front view of the mandible showing shortening on left side and midline displacement. Right: Left mandible after oblique osteotomy and direct wiring of fragments

mandible about 3/16 inch apart. The osteotomy was completed by connecting these drill holes with a large fissure bur.

The long fragment of the mandible was slid forward on this diagonal osteotomy and overcorrected, bringing the chin in the midline. The two bone fragments were wired directly with stainless steel wire (Fig. 2, right).

About two months later, contouring bone grafts taken from the crest of the ilium were applied.

The mandible at its lower border was laid bare from the angle on the left side well past the midline. The bone grafts were held in position to the mandible by circumferential wiring passing through the body of the mandible (Fig. 3). The object of the bone grafts was to obtain a sculptured symmetry of the jaw and chin (Fig. 1, right).—*804 West Seventy-Ninth Street.*

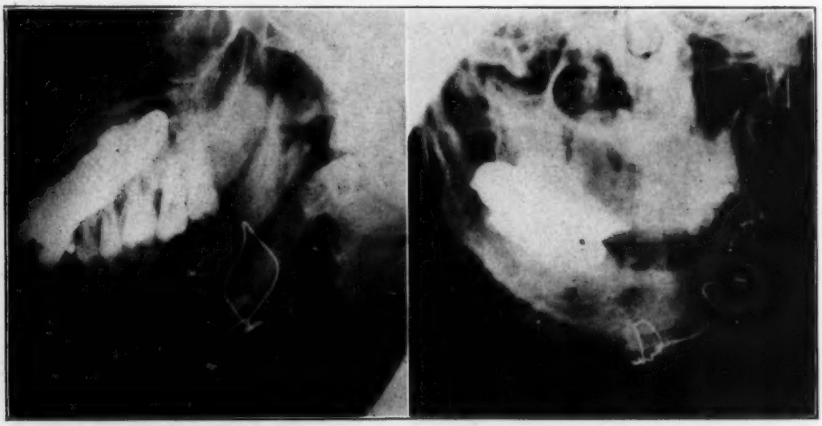

Fig. 3.—Roentgenograms. Left: Lateral view showing sculpturing of bone grafts to restore angle contour. Right: Front view of bone grafts on left side and mental region

PERIMANDIBULAR OSSIFICATION OF POSSIBLE TRAUMATIC ORIGIN: REPORT OF CASE

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New York

After many years of experience in the field of oral surgery, one still encounters conditions which have not been recorded previously in the literature. The case to be presented is one of these, for the author can recall no previous report on the occurrence in the literature.

History.—A girl, aged 12 years, came to the oral surgery clinic of the Hospital for Joint Diseases on March 18, 1947, with a pronounced swelling of the left side of the mandible. She appeared to be well developed and well nourished and gave no history of former ailments of any consequence. Familial history and general medical examination, including clinical laboratory studies, disclosed nothing relevant. Although the swelling of the jaw had been present for some weeks, there was no pain at any time, nor was there elevation of temperature. Medical advice was sought because the swelling had been increasing in size.

Examination.—The surface swelling extended from the mental region to the inferior border of the zygoma and well below the angle of the mandible (Fig. 1). The superficial soft tissues were free from inflammation and glided smoothly over the underlying indurated swollen mass. Upon deep palpation the mass was found to be hard, painless and not movable. It was distinctly osseous in character and firmly adherent to the normal bone. It was smooth in outline and there were no soft or fluctuant points.

Intra-orally the mucous membrane was normal in all respects; the only local abnormality was a missing first molar. By deep palpation beyond the anterior border of the ascending ramus, the osseous mass could be discerned easily.

The only possible pertinent past history was that the lower left first molar had been removed about three months previously because of caries. As is a common occurrence, the ex-

Fig. 1.—Two aspects of the swelling in the mandible at the time of the first examination

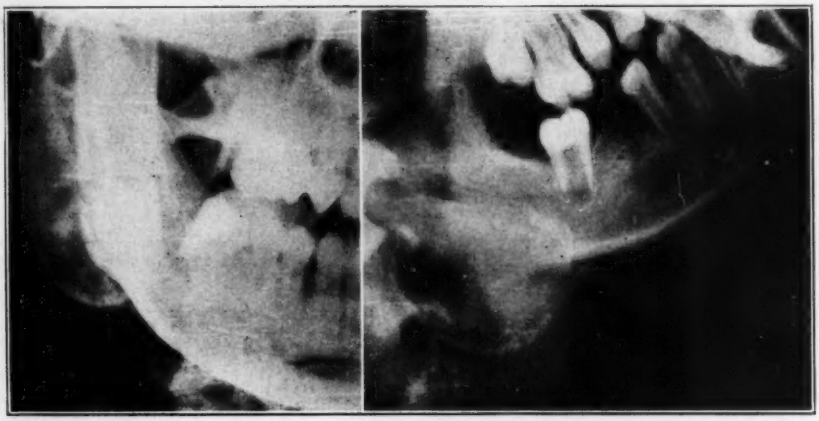

Fig. 2.—Roentgenograms showing osseous proliferation. The extent of new bone formation and the intact normal bone are apparent. Left: Anteroposterior view. Right: Lateral view

traction was followed by moderate swelling and trismus which disappeared gradually. In spite of the local difficulty, the patient was able to open her mouth freely at the time she came to the clinic for advice about the large perimandibular swelling which had developed after the extraction.

The roentgenograms of this swelling showed an osseous mass which gave the impression of being pasted or plastered to the major part of the ascending ramus. It extended well below the angle of the mandible. The normal unimpaired bone could be distinguished clearly (Fig. 2).

Diagnosis.—A consultation was held with the hospital's pathologist (Dr. Henry L. Jaffe). The tentative diagnosis was fibrous osteoma or ossifying hematoma—more likely the latter. It was felt certain that, in any event, the lesion was benign. This opinion was supported especially by the roentgenographic finding that the basic bone was intact in substance.

As the swelling was increasing perceptibly in size, it was decided to take a biopsy specimen. The question arose of whether the biopsy should be taken through an external incision or intra-orally. In order to obtain more precisely the specimen desired, an extra-oral approach was decided upon. The advisability of removing all the newly formed tissue, which could be differentiated easily in the roentgenograms, also was considered.

Operation.—On April 15, 1947, the patient was hospitalized. The following day, with the aid of general anesthesia, an incision of about 2½ inches was made below the lowest line of the curvature of the swelling. With sharp and blunt dissection the border of the bony mass was exposed. The proliferated osseous tissue was cut easily with hand pressure chisels and even a knife blade. A block of this tissue, about 3 cm. in diameter, was excised. The wound was closed with interrupted sutures for primary union.

Although strict surgical technic had been observed in the operation, the wound did not heal by first intention. Local infection ensued, and the lips of the wound broke down. Cicatrization was progressive but slow. There were no systemic effects. After twenty days the wound healed and the patient was dismissed with instruction to return for further observation.

Pathologic Report.—"The specimen consists of fragments of loose osseous tissue. Microscopic findings show extensive subperiosteal new bone formation and also evidences of a low grade infection." A bacteriologic specimen taken from the exposed bone surface at the operation was negative (Fig. 3).

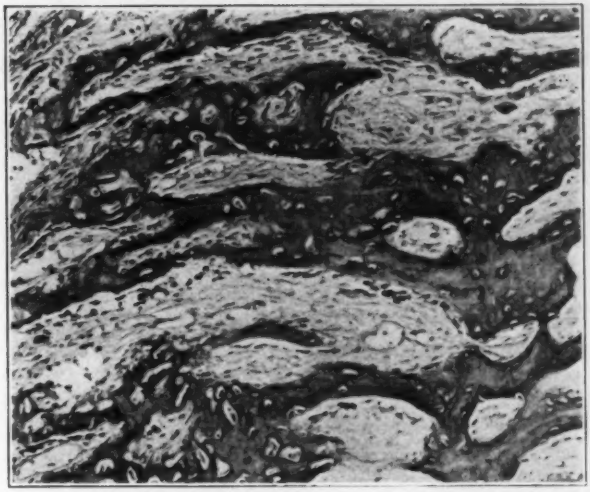

Fig. 3.—Photomicrograph of the specimen excised shows the histologic structure of the bone

Course.—Soon after the operation, even while the patient was under postoperative treatment, a recession of the swelling was noted. Because of this retrograde, resolving process which was not explained, further surgical interference was deferred for the time being.

There was a summer recess. After three months, the patient was recalled. At this time her facial outline was entirely normal. The roentgenogram showed that the abnormal osseous formation had been resorbed completely (Fig. 4). The incision which had been made for the biopsy healed with a keloid, however (Fig. 5). The patient was transferred for treatment with physical therapy in the hope of resorption of this keloid.

Comment.—The nature and cause of this newly formed osseous tissue was baffling. It should be stressed that the first molar had been removed. The roentgenogram (Fig. 4) shows that

Fig. 4.—Roentgenograms of normal mandible. Left: Complete resorption of the osteoid formation, a normal outline and texture of the mandible (May 11, 1948). Right: Bone deposits shown in Figure 2 have disappeared

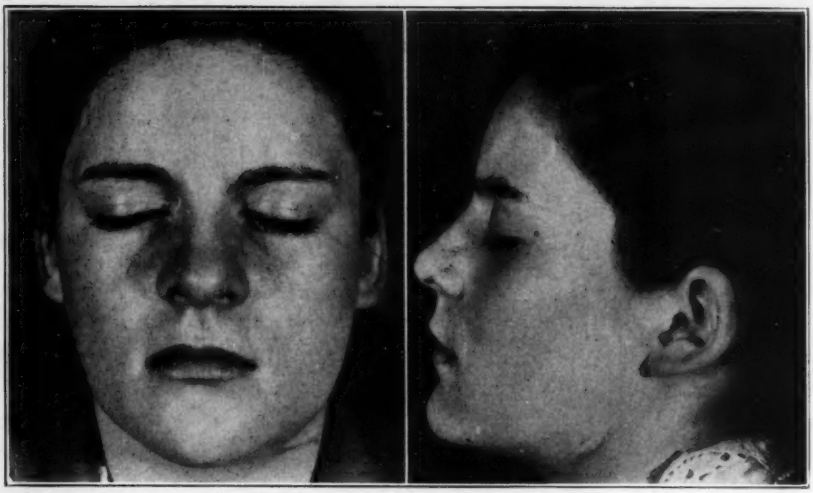

Fig. 5.—Photographs of the patient taken May 11, 1948. Left: Front view. Right: Side view showing cicatricial keloid which is shrinking

this tooth socket was filled completely with new bone. Was the removal of this tooth or some other overlooked trauma responsible for this lesion? It is difficult to conceive that the removal of a lower first molar, however crudely done, would induce such widespread subperiosteal extravasation as to cover the entire outer surface, and more, of the ascending ramus.

Could surgery of the biopsy section have induced a resolution of the whole process? That this tissue formation was subperiosteal is suggested strongly by the fact that function was not impaired. The absence of pain in the lesion indicated that no muscles were involved.—*654 Madison Avenue.*

Carcinoma of the Lip, Preliminary Analysis of 330 Cases with Followup of From 3 to 36 Years. Joseph L. Bernier and Mardelle Clark, in *Mil. Surg.* 102:474 (June) 1948.

Conclusions

From these limited data and other published reports, it is reasonable to assume:

First: That lip carcinoma, which represents approximately 5 per cent of all carcinomas occurring in the oral regions, if detected and treated early, will show a high percentage of favorable results.

Second: Although this series indicates that carcinoma of the lip may occur more frequently in young men than is popularly supposed, it is unlikely that it represents a high rate of occurrence in relation to the general population.

Third: As a rule lip carcinomas are of low-grade malignancy and are quite amenable to surgical removal which is, in most instances, preferable to radiation.

UMI

COMPLETE CORONAL RESORPTION OF A TOOTH IN A DENTIGEROUS CYST: REPORT OF CASE

James M. Helzberg, D.D.S., M.S.D.,
Kansas City, Mo.

A BOY, aged 9 years, presented himself for routine dental checkup. Examination disclosed an apparently healthy mouth with no caries or gingival disorders. However, it was observed that the lower left permanent first molar was missing from the arch. The other permanent first molars had erupted and were in good condition and occlusion.

Examination.—Roentgenograms were made of the lower left molar region to determine a possible cause for the absence of this molar. The roentgenograms revealed a radiolucent area in the body of the mandible in this region (Figs. 1 and 2, left). This large area (2 cm. in diameter) was outlined clearly by normal bone. In the center of the area were two molar roots, and at their occlusal surface a radiopaque image having a "cotton wool" appearance was evident.

Further clinical examination revealed that the buccal plate of bone had been distended in this region, creating a slight swelling. Upon palpation the buccal bone exhibited egg-shell consistency. The area was covered with normal mucous membrane, and a sinus or communication with the oral cavity could not be demonstrated. The area was aspirated with a hypodermic syringe, and it was found that the cavity was filled with a clear, colorless, serous fluid.

These clinical and roentgenographic findings are consistent with the picture seen in a dentigerous cyst, and this preoperative diagnosis was made.

Operation.—Premedication of 1½ grains of pentobarbital sodium was administered. The operation was performed under local anesthesia. A 3 cc. incision was made along the crest of the ridge, the mucoperiosteum was retracted and a flap was formed in this manner. No bone was present over the occlusal surface of this cavity, and that in the buccal direction was extremely thin. In order to obtain better access, approximately 0.5 cc. of the occlusal portion of the buccal plate was removed with rongeur forceps.

It was impossible to shell out the surrounding membrane completely because of its thinness, toughness and adherence. This tissue lining the bony cavity was removed with a curet.

Postoperative Care.—Three hundred thousand units of penicillin were dusted over the surface of the wound and the area was packed with petrolatum gauze. The mucoperiosteal flap was sutured in place. This dressing was removed forty-eight hours after the operation and replaced by a gauze drain. The drain was changed and the wound irrigated daily for four days. The wound healed uneventfully, and the patient experienced a minimum of postoperative swelling and discomfort (Fig. 2, center and right). Paresthesia did not follow the operation.

Pathologic Report.—The membrane was sent for examination. The pathologist's report stated: ". . . section through the small pieces of tissue show varying amount of bone spicules in a state of calcification. There are some areas of osteoid or noncalcified tissue. The main portion of the section shows a fibrous tissue with many blood vessels and a scant round cell infiltration. In several places, we see fragments of a squamous epithelial cell lining. These findings are consistent with a dentigerous cyst."

The two roots subsequently were decalcified and sectioned. Microscopic examination disclosed that the dentin at the occlusal surface of the roots was undergoing resorption (Figs. 3, 4, 5). The presence of Howship's lacunae and a few scattered giant cells having the appearance of osteoclasts attested to this. It cannot be concluded that the entire crown has been resorbed because the possibility of incomplete formation of the crown must be considered. But it is certain that resorption has accounted for the loss of some tooth substance and that the

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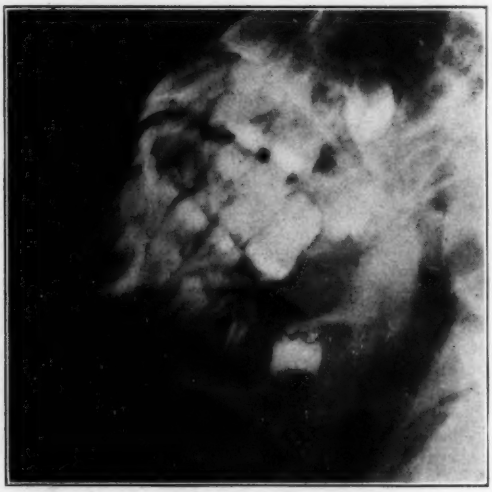


Fig. 1.—Preoperative lateral jaw roentgenogram showing radiolucent area in body of mandible

destruction definitely is not caused by caries. If caries had caused this process, bacteria would be seen in the dentinal tubules. The dentin which has been resorbed is being beplaced by fibrous tissue and at many places by bone. The pulp was normal. The irregular radiopaque shadow seen in the roentgenograms at the occlusal surface of the roots is spongy bone.

In 1941 Isaacson[1] reported a case in which there were two large radiolucent areas present in the occlusal surface of a completely embedded lower bicuspid. These cavities were interpreted to be the result of caries and subsequently were filled after the tooth had erupted. A histologic examination was not made in order to ascertain definitely if this destruction was caused by caries or resorption. It is difficult to understand how caries could occur in a completely embedded tooth in which there was no communication with the oral fluids.

Skillen,[2] in the same year, described a case of a completely embedded lower third molar

1. Isaacson, S., Intrafollicular Caries. *D. Items Int.* 63:184 (February) 1941.
2. Skillen, W. G., So-Called "Intra-Follicular Caries." *Illinois D. J.* 10:307 (August) 1941.

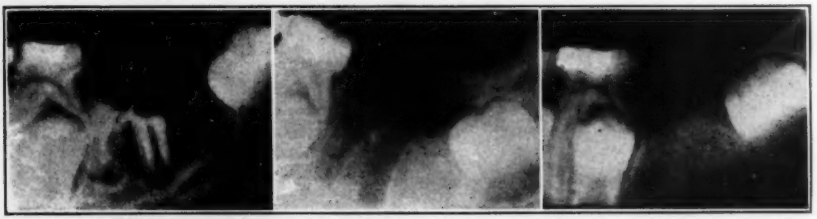
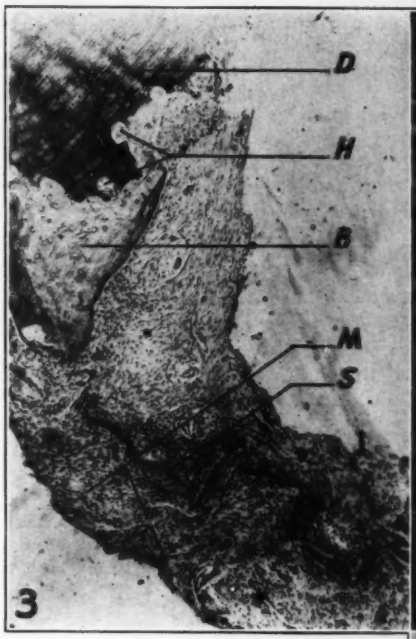


Fig. 2.—Left: Preoperative roentgenogram showing two molar roots in radiolucent area. Center: Postoperative roentgenogram after removal of 0.5 cc. of occlusal portion of buccal plate and curettage of tissue lining bony cavity. Right: Roentgenogram taken five months after operation showing complete regeneration of bone in region



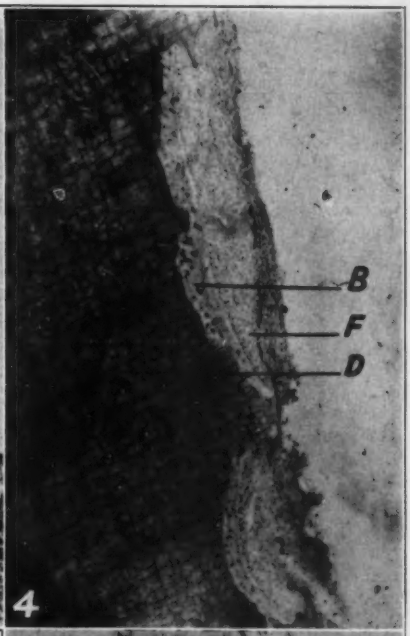
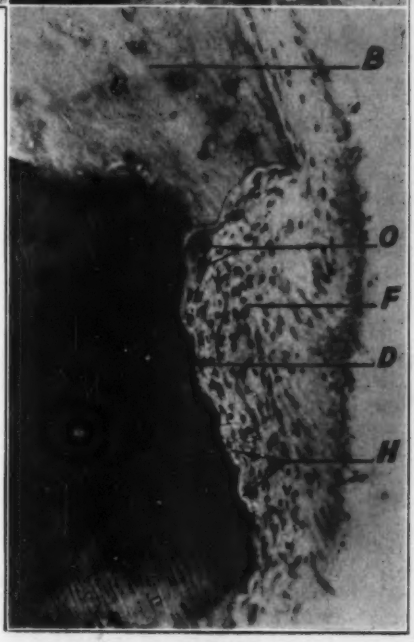


Fig. 3.—Photomicrograph of occlusal area. D: Dentin. H: Howship's lacunae. B: Bone repair of resorbed dentin on occlusal surface. M: Fibrous tissue membrane. S: Bone spicule

Fig. 4.—Photomicrograph of root area. B: Bone repair of resorbed dentin. F: Fibrous tissue. D: Dentin

Fig. 5.—High power photomicrograph. B: Bone repair of resorbed dentin. O: Osteoclasts. F: Fibrous tissue. D: Dentin. H: Howship's lacunae



in which there was a radiolucent area in the dentin under an intact enamel cap. This tooth was removed, decalcified and sectioned. Microscopic examination disclosed that the radiolucency was not caused by caries but rather was the result of resorption. The present case is unusual in that it presents a completely embedded tooth in which the entire crown is missing. This extensive loss of tooth substance seems to be the result of resorption.

The underlying etiology of this resorption and proliferation is unknown. The inflammatory process which was present could have caused proliferation and cystic degeneration of the dental follicle as well as initiated resorption of the embedded tooth, giving rise to a partially destroyed tooth present in a dentigerous cyst. Neither the patient nor his parents could recall any history of trauma or other disturbance in the region that might have caused such an inflammatory reaction.—*1102 Professional Building.*

Congenital Cysts and Fistulae About the Head and Neck. Charles D. Blassingame, in *Ann. Otol., Rhin. & Laryng.* 56: June, 1947.

Summary

General agreement upon the origin of the thyroglossal duct has resulted in standardized criteria of diagnosis and treatment of cysts and fistulae having their origin from this duct.

The embryologic background of lateral cysts and fistulae of the neck influence the clinical or anatomic and technical problems associated with them. Wide divergence of views in regard to their derivation has created much uncertainty and confusion as to their surgical management.

Early observers believed that lateral cysts and fistulae of the neck were derived from the branchial apparatus. In 1913, Wenglowski published a monograph disputing the possibility of the branchial origin of lateral cysts and fistulae, presenting evidence instead that these anomalies have their origin from persistence of parts or all of the thymus corridor.

A case is reported illustrating a complete lateral fistula of the neck having its internal opening in the right tonsil at the upper pole, and its external opening at the anterior border of the right sternomastoid muscle near the sternal notch. In this case, the tonsil was removed with the fistula intact. We were unable to find in the literature another case in which a similar procedure was used.

Mixed Tumors of the Parotid Gland. J. D. Martin, in *New Orleans M. & S. J.* 99:99 (September) 1946.

Mixed tumors of the parotid gland are complex in structure, with all cell types usually being represented; they may be attached or in the substance of the gland and appear as nodules or masses in the region of the parotid gland or below or behind the ear. Tumors are usually firm, and encapsulated; larger tumors are found to fuse with the connective tissue of the gland. Mixed tumors occur most often in the third and fourth decades, but the age at which they appear is not related to their malignancy. Malignant growths are extremely painful and enlarge rapidly; ulceration occurs when growth interferes with circulation. The survival rate for those with malignant disease is two years after operation, malignant degeneration occurring more often after excision.

Treatment should be delayed pending careful observation, the choice being between surgery and irradiation, or a combination of the two. Results to date indicate irradiation is of benefit only palliatively and preoperatively, to lessen the risk of rupture during operation. Excision involves the danger of great facial paralysis from section or paralysis of the facial nerve, which runs forward from the stylomastoid foramen in the substance of the gland. Surgery is indicated when the discomfort of the patient is extreme and when the tumor is malignant. Recurrences are usually local, but distant metastasis may occur. 14 references. 2 figures.—*Quart. Rev. Otorhinolaryng. & Br.-Esoph.* 6: March, 1947.

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BOOK REVIEW

Bone and Bones. By Joseph P. Weinmann, M.D., College of Dentistry, University of Illinois, and Harry Sicher, M.D., School of Dentistry, Loyola University, Chicago. 464 pages, illustrated. Cloth. Price $10. St. Louis: C. V. Mosby Company.

In this outstanding book, the authors have drawn upon a large number of distinguished scientists for specimens which, added to the material from the Department of Research, Loyola University School of Dentistry, provide the finest of illustrations to accompany an admirable text.

Part I covers the normal structure and growth of bone and bones. In Chapter I the structural elements of bone tissue, the arrangements of these elements, the processes of development and growth of bone tissue, calcification, regeneration, and reconstruction provide the foundation for an adequate study of diseases and tumors of bone. In Chapter II, the details of the structural elements and development of the bones of the skeleton are described, including transverse and longitudinal growth, modeling resorption, and growth of membranous bone. Of great interest and importance to the oral surgeon is the intimate discussion of the growth of the facial skeleton, the maxilla and mandible, and the accessory sinuses of the nose. The section on facial growth and tooth eruption and the developmental mechanism of impactions contains much of value. The final section of this chapter includes most interesting and informative material on the functional adaptation of bones, and the reaction of bone to pressure and traction.

Part II is made up of nine chapters on the pathology of bone and bones. Developmental disturbances of bone tissue (osteogenesis imperfecta and osteopetrosis), and the generalized developmental disturbances of the bones (chondrodystrophia fetalis, hereditary multiple exostosis, and cleidocranial dysostosis) are included in Chapter III, serving as a ready reference text for the oral surgeon in these unusual conditions with dental and facial implications. Chapter IV covers adaptational deformities of the skeleton with a fundamental discussion of biologic plasticity of bone, including the deformities of the skeleton in severe cases of rickets, osteomalacia, and Recklinghausen's disease. Changes resulting from malunited fractures, ankylosis, permanent dislocations, increased function, and lack of function provide most interesting and informative reading. Of particular importance is the detailed description of atrophic changes in the jaw that take place when teeth no longer function and when teeth are lost. The processes of repair of a tooth socket consisting of a regenerative phase followed by reconstruction, which later becomes subject to disuse atrophic resorption in greater or less degree in accordance with the functional stimulation afforded by partial or complete dentures, should be understood fully by every oral surgeon.

The influence of endocrine glands (intrinsic hormones—pituitary, thyroid, male and female sex glands, and the parathyroid gland) upon bone and bones is treated extensively in Chapter V. Of great interest and value is the section of hyperparathyroidism, which includes a detailed discussion of the pathology of osteitis fibrosa cystica. Included is the description of benign giant cell nodes, bone cysts, and an analytic discussion of the various concepts regarding the development and pathology of giant cell tumors—a subject of every-day value to the oral surgeon.

Chapter VI—the influence of vitamins (extrinsic hormones) upon bone and bones—provides the reader with an effective knowledge of this subject so frequently discussed without adequate scientific and clinical background. The same may be said of Chapter VII on the effect of minerals on bone and bones—both informative and interesting chapters.

Chapter VIII—healing of bones, fractures, and postextraction sockets—provides the oral surgeon with a well illustrated and detailed account of bone repair.

Chapter IX—necrosis of bone and the inflammations of bones—is a fundamental pathologic presentation of this highly important subject and worthy of intensive study.

Chapter X—tumors of the skeleton—presents an outstanding discussion of tumors involving bone, primary and secondary, benign, and malignant. In a lengthy discussion of anaplasia, the authors challenge the assumption that some cells of a tissue may remain in an undifferentiated embryonic state later to become foci of tumors. They distinguish clearly between this hypothesis and the examples where remnants of embryonic organs or structures fail to disappear in the normal course of development and may produce tumors, such as branchial cysts.

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The authors present a classification of primary tumors of the skeleton on the basis of two types of tissue of origin: supporting tissues (including connective tissue, cartilage and bone osteoblasts) and accessory tissues (including the blood vessels, adipose tissue, bone marrow, blood forming elements and the reticulum). Secondary metastatic tumors are epithelial or mesodermal.

The reaction of bone to the growth of tumors provides most interesting and instructive reading of great value to the oral surgeon for a better understanding of the behavior of tumors of the jaws. Well selected photomicrographs accompany the intimate discussion of the pathology of the various types of tumors, both benign and malignant. In the section on ossifying fibroma, the authors do not mention nor discuss fibrous dysplasia of bone, a variant or stage of ossifying fibromas. In this they disregard the trend of research and the growing literature of this subject.

The final chapter, XI, is devoted to Paget's bone disease—osteitis deformans. Attempts to classify this entity as either a tumor or an inflammatory disease have been unsuccessful. Facial involvement in Paget's disease is relatively uncommon and, if it is extensive, produces the condition known as leontiasis ossea.

Thirty pages of references are a most valuable addition to the text, and the index is well arranged.

This book is a summary of the world's literature on the subject, and is essentially a complete discussion of the anatomy, physiology and pathology of bone. Future editions will afford the authors the opportunity to include material to improve the text from the standpoint of applied clinical oral surgical pathology. Some of the authors' conclusions may be modified or changed in the light of future research, but in its present edition this invaluable text should be a desk manual for every oral surgeon.—*Carl W. Waldron, M.D., D.D.S.*

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